REPORT

OF THE

NEW YORK STATE VETERINARY COLLEGE

AT CORNELL UNIVERSITY

For the Year 1915-1916

TRANSMITTED TO THE LEGISLATURE MAY 10, 1917

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REPORT OF THE NEW YORK STATE VETERINARY COLLEGE

STATE OF NEW YORK — EXECUTIVE CHAMBER

ALBANY, *May* 10, 1917

TO THE LEGISLATURE:

I have the honor to transmit herewith the Annual Report of the New York State Veterinary College at Cornell University for the academic year 1915–1916.

CHARLES S. WHITMAN.

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REPORT

JANUARY 4, 1917.

The Honorable CHARLES S. WHITMAN, *Governor of the State of New York, Albany, N. Y.:*

SIR.—The Act of the Legislature of the State of New York approved May 22, 1897 (chapter 689 of the Laws of 1897), providing for the administration of the New York State Veterinary College at Cornell University, contains the following provisions:

"The said university shall expend such moneys and use such property of the State in administering such veterinary college, and shall report to the governor during the month of January in each year a detailed statement of such expenditures and of the general operations of said veterinary college."

In accordance with this mandate I have the honor to submit in behalf of Cornell University the accompanying report of the operations of the New York State Veterinary College for the year 1915–16.

The following report of the Dean of the College, supplemented by the special reports of the heads of the several departments, gives a detailed and instructive account of the teaching and research work carried on by the College during the past year. The successful service performed by the College and its appreciation by the veterinary profession and the stock-owners of the State render it necessary to make some extensions of the buildings and equipment and some enlargement and improvement of the staff devoted to instruction and investigation. And the State has a direct financial interest in making the appropriations necessary for this purpose. For, as Dean Moore has explained, we are dependent for the prevention of infectious diseases, especially those of a chronic nature, upon the skill and training and efficiency of our veterinary practitioners. In proportion, therefore, as appropriations are made to improve the training of veterinary practi-

tioners will the amount of money required to carry out the provisions of the diseased animal acts of the State be reduced. Either the State must train its veterinarians so thoroughly that they can adequately aid the owners to protect for themselves their flocks and herds, or the State is liable for the payment of hundreds of thousands of dollars annually in indemnities for diseased animals and the expenses incurred in the enforcement of sanitary laws.

This subject is presented with admirable force and clearness in the Joint Report on Foods and Markets which has just been presented to the Legislature by Governor Whitman's Market Commission, Mayor Mitchel's Food Supply Committee and the Wicks Legislative Committee. In the sixth section of that report these joint committees, referring to the part played by agricultural and veterinary colleges in the production of food supplies, insist that it is good State policy to provide for such institutions the best scientific experts and to furnish them with all the necessary facilities in the way of buildings and equipment, and then proceed as follows:

"Other countries are far ahead of us in this respect. For instance, during the last five years the little country of Norway, with a cattle population of 1,100,000, expended $650,000 for a new veterinary college and equipment; while New York State, with a cattle population of 2,500,000, has expended less than $400,000 on its veterinary college and equipment during the last twenty years. Belgium recently rebuilt her college at a cost of $1,000,000; Holland has a college that cost nearly $1,000,000, and Germany's many activities in this direction are well known.

"The value of the live stock of this State, as shown by the United States census of 1910, was $246,000,000, with an annual loss from disease of about $25,000,000. With a veterinary service developed to the degree of efficiency that has been attained in European countries this loss could undoubtedly be reduced at least 50 per cent. The State should also appropriate more money, through its veterinary college, for the study of the causes and prevention of animal diseases and for the better preparation of veterinarians who are to become the advisers of live stock owners and through whose knowledge and advice the startling inroads of

animal diseases upon the production of meat and dairy products can be prevented, and the development, upon a sound basis, of the stock raising industry of the State can be promoted."

I endorse every word of this statement from the report of the three distinguished committees who represent the Governor and Legislature of the State and the administration of the city of New York and earnestly commend it as a basis of action to the Governor and Legislature in the winter of 1917 in making appropriations for the State Veterinary College at Cornell University.

I have the honor to be

Your obedient servant,

JACOB GOULD SCHURMAN,
President of Cornell University.

REPORT OF THE DIRECTOR

December 30, 1916.

President J. G. SCHURMAN:

SIR.— I have the honor to submit herewith a report of the New York State Veterinary College at Cornell University for the nine months ending June 30, 1916. This shorter period is due to the change in the beginning of the fiscal year from October 1 to July 1.

The academic year which ended in June, 1916, saw the close of the three-year curriculum in this College, and the opening of the University in October witnessed the beginning of the four-year course of instruction in veterinary medicine. This, educationally, is an advance worthy of special mention. It was the ambition of the first director of this College, Dr. James Law, that the preparation for the practice of veterinary medicine should be on at least an equal footing with that of other learned professions. He recognized that those engaged in supplying dairy products, meat, leather and wool to our people would eventually be dependent for their success, because of the diseases of animals, upon men trained in the sciences that make up the veterinary curriculum.

The qualifications necessary to enable veterinarians to render valuable assistance in combating animal diseases can be attained only by adequate training in the required sciences and careful instruction in their practical application. It is because of the necessity of personal supervision and direction of the student in clinical as well as in laboratory work that the cost of maintaining a veterinary college is relatively high, but without such teaching the course would be inadequate and the existence of the college unjustified. Colleges of human medicine have recognized this principle and planned their courses accordingly.

The problem for this College is to train veterinary students not only in the nature and treatment of many common diseases of several species of animals but also in the methods for preventing the spread of the infectious maladies. These are costing the State

millions of dollars annually and they should be prevented. It is the practicing veterinarian, consulted by the owner of infected animals, who largely determines the fate of the herd. If he lacks the necessary knowledge, and consequently does not give proper advice, the loss is likely to be heavy. If he is qualified by the possession of adequate technical knowledge the procedure indicated will be followed and the loss minimized. The prevention of the infectious diseases, especially those of a chronic nature, depends so largely upon the efficiency of the practitioners that too much emphasis cannot be placed upon proper instruction. The amount of money required to carry out the provisions of the diseased animal acts of the State will be reduced in proportion to that expended to properly prepare its practitioners. The State is confronted with the proposition of either preparing its veterinarians so that they can adequately aid the owners to protect for themselves their flocks and herds, or paying hundreds of thousands of dollars annually in indemnities for diseased animals and expenses incurred in the enforcement of sanitary laws.

The present high prices of live stock, meat and dairy products are, to an appreciable degree, due to the losses from the diseases of animals. Last year the Federal meat inspection service condemned 299,958 whole carcasses and 644,688 parts of carcasses for disease. The State and city inspectors condemned many thousand more. While enormous, these losses are very little compared with those caused by preventable diseases such as abortion, sterility and tuberculosis in the dairy herds of the State. These troubles are technically very difficult to overcome, and not until adequate provision is made to combat them will the losses they occasion be reduced to a minimum. It is the recognition of more efficient professional education as the only solution of the live stock sanitary problems of the State, which economically involve millions of dollars annually, that impels us to urge more liberal support for the veterinary college, better preliminary preparation and a longer and more thorough training of veterinary students.

The number of new students that applied for admission at the opening of the four-year course was nearly up to the normal registration under the three-year curriculum. This argues well for the wisdom of the advanced step taken, and also it exemplifies

the appreciation of the public in having the preparation for veterinarians on a basis more in keeping with their duties and responsibilities. The importance of the work that veterinarians alone are qualified to do is beginning to receive the recognition it deserves from both the economic and sanitary points of view. This has increased the demand for technically well equipped and efficient veterinarians.

The new department established in 1914 for the investigation of the diseases of breeding cattle and those of the new born was successful both in gaining new information and arousing interest in the subject. An appropriation of $15,000 was secured from the Legislature for this purpose and to erect suitable buildings for the experimental work. During the unavoidable delay in the erection of the buildings certain investigations are being carried out in a few large herds whose owners have very kindly placed them at our disposal for the purpose of study and for applying certain methods for the control of infectious abortion and sterility. By this means information will be obtained that should be of assistance in eventually formulating methods of procedure that can be recommended to the practitioner for giving relief from the enormous losses now being sustained because of these affections. The securing of accurate data concerning the nature of these troubles and the finding of methods for their prevention are the problems before this new department. Owing to the great advance in the cost of building, we find that but a small part of the appropriation will be left, after the necessary construction, for continuing experimental work. It will be necessary because of this unusual condition to request a small additional appropriation to continue this work next year.

The number of matriculated undergraduate students was larger, the quality of teaching has been better, and the clinical material more abundant than in any previous year. The research work has been fully as productive as heretofore. The efforts of the College have followed the regular channels of teaching, preparing biologic diagnostic agents and anti-hog cholera serum, and research. In the appendix will be found a detailed account of the animals treated in the clinics and a number of reports that will indicate the character of work that is being done. With the small faculty,

the large number of subjects to be taught and the very moderate appropriations, the operations of the College beyond its first duty of giving instruction must necessarily be restricted far more than the needs of the profession require or the losses sustained from disease demand. To increase the work means larger appropriations.

During the year the College has received a very interesting collection of specimens of bone diseases, the skull and pelvic bones of an elephant and several surgical instruments of much historical interest from Dr. L. McLean of Brooklyn, N. Y. Dr. Benjamin Pierce of Springfield, Mass., presented the College with a beautiful etching of Professor Thomas Walley, formerly of Dick Veterinary College, Edinburgh, Scotland. Dr. Walley is known to American veterinarians through his excellent work on meat inspection. He was a pioneer in that field of veterinary service. We acknowledge with thanks these contributions of instructive and valuable material.

Faculty

There were few changes in the faculty. This explains to a large extent the increased efficiency in teaching. At the close of the year, however, Dr. L. A. Wright, instructor in medicine, accepted a position at nearly twice the salary he received here in the new veterinary college in Texas. Dr. J. F. Shigley resigned as instructor in surgery to accept a much better position in South Dakota. Tempting offers are before other members of the faculty, but we hope they will not be accepted. New York needs the service of these men who have a true scientific attitude and devotion to teaching and research.

The University furnished in other colleges instruction to the veterinary students in animal husbandry, chemistry, embryology and histology. The personnel of the veterinary faculty is appended.

Jacob Gould Schurman, president of the University.
James Law, emeritus professor of medicine.

Department of Anatomy

Dr. Grant S. Hopkins, professor of anatomy.
Dr. Earl Sunderville, assistant professor of anatomy.
Dr. Howard E. Johnson, instructor.
Mr. Erwin V. Moore, assistant.

Department of Materia Medica

Dr. Howard J. Milks, professor of therapeutics and small animal clinic.
Dr. W. E. Muldoon, instructor.

Department of Medicine

Dr. Dennie H. Udall, professor of veterinary medicine and hygiene and superintendent of the ambulatory clinic.
Dr. F. F. Koenig, assistant professor of medicine.
Dr. L. H. Wright, instructor.

Department of Pathology and Bacteriology

Dr. V. A. Moore, professor of comparative pathology and bacteriology and dean of the College.
Dr. Samuel H. Burnett, professor of comparative pathology.
Dr. Clifford P. Fitch, assistant professor of bacteriology.
Dr. Earl M. Pickens, assistant professor in diagnosis.
Dr. S. A. Goldberg, assistant in pathology.
Mr. William A. Billings, assistant in diagnosis.
Mr. Joseph B. Latshaw, B. S. A., student assistant.

Department of Physiology

Dr. Pierre A. Fish, professor of veterinary physiology and secretary of the faculty.
Dr. C. E. Hayden, assistant professor of physiology.

Department of Obstetrics and Research in the Diseases of Breeding Animals

Dr. W. L. Williams, professor and head of the department.
Miss Ethel Williams, student assistant.

Department of Surgery

Dr. J. N. Frost, assistant professor of veterinary surgery.
Dr. James F. Shigley, instructor.

Veterinary Experiment Station

Dr. Raymond R. Birch, superintendent of the station.

Farriery

Mr. Henry Asmus, assistant professor of horseshoeing.

Library

Miss Frances B. van Zandt, librarian of the Roswell P. Flower Library.

Business Office

Miss Helena H. Haight, clerk and bookkeeper.
Miss Lulu M. Williams, stenographer and office secretary.

Engineers

Mr. Archibald Wilson, engineer.
Mr. Charles Savercool, assistant engineer.

Thirteen regular employees were required to do the necessary work in the care of buildings, patients in the hospitals, and experimental animals and as attendants in the different departments and laboratories. In addition to these, special work necessitated for short periods of time the employment by the day or hour of a few men.

The experiments and research work that are being done by different departments and which involve the use of animals are conducted at the veterinary experiment station. The superintendent has the general care of these animals and assists members of the faculty as much as possible in their experimental work. He has charge of the preparation of the anti-hog cholera serum which is made at the experiment station. In addition to this work, Dr. Birch has made some important discoveries, as the result of careful experimentation, on the means by which hog cholera is disseminated. The station has been and still is a great asset to the college in giving a place and affording facilities for numerous small experiments necessary to ascertain the facts in connection with many undetermined or disputed points that are constantly being encountered in teaching veterinary medicine.

At the time the college was opened in 1896 there was a great need in the State for a laboratory for diagnosis of animal diseases and where the preparation of biologic diagnostic agents and anthrax vaccine could be made. Realizing this need, the college started a laboratory for this work, which has proven to be of great assistance to the veterinarians and live stock owners as well as to the State Department of Agriculture. For the twenty years of its existence this college has furnished, without charge, the tuberculin, mallein and anthrax vaccine required by the State department in administering the laws pertaining to animal diseases. It has also furnished the veterinarians of the State with these products at the cost of material for their preparation. It has also made the diagnosis for its veterinarians where laboratory facilities were required. The gradual increase in the quantity of work of this kind has enabled the college to render much service to the State which in other commonwealths is provided for by special appropriations and separate laboratories. It is difficult to estimate the value to the public of this work. A prompt diagnosis of an infectious disease has often prevented its further spread. The work, however, is becoming a heavy tax upon the resources of the college, and, because of this service, more liberal appropriations for maintenance are justly requested.

The diagnosis work has a teaching significance of much importance. It affords material for the actual demonstration of laboratory methods in diagnosis. This is of unquestioned assistance in the practical teaching of this subject. Valuable material for research is often obtained in this way.

The farriery and the employment of an expert horseshoer have made it possible to give a course of practical instruction in horseshoeing to the students in the senior class. As many forms of lameness can be corrected by proper shoeing, it is most desirable to have such facilities in connection with the consulting clinic.

The College also offers six-weeks courses in horseshoeing for practical horseshoers. These are given every two months, beginning with January. The number of horseshoers who have taken this course is smaller than was expected; however, it is still believed that this opportunity for valuable instruction will be more appreciated in the future and that these courses will prove

to be of much direct assistance to the horseshoers and indirectly to the horse owners of the State.

Library

The library contains 5,168 volumes, of which 4,903 volumes belong to the Flower Library. During the year 254 books have been added, of which 176 belong to the Flower Library and seventy-eight to the State. The library receives ninety-one periodicals on veterinary medicine and closely allied subjects, together with a few journals on agriculture, animal breeding and human medicine. These are all available for students as well as faculty. A trained librarian is constantly in charge. The usefulness of this now excellent veterinary library is restricted because of lack of space. It is planned to add, when constructed, the second floor of the south wing to the room now occupied by the library. With this addition the library facilities will be very satisfactory.

Students

As this was the last year that students could enter on a three-year course, there was naturally a large increase in the number of new matriculates. There were seventy-seven new students who registered in September, 1915, and thirteen others who registered late or in February, 1916, making a total enrollment of ninety freshmen. There were forty-one second-year students, twenty-nine seniors, five graduate students and three in the practitioners' course. The practitioners' course has been designated as a proper one under which to enroll veterinarians of the State who wish to study for a short or longer time at the College. They are allowed to attend any lectures or clinics they desire, and are given such special instruction as possible. The average enrollment of new students for the five years preceding 1915 was forty-eight. The registration of new students in October, 1916, was thirty-six. This shows a small reduction in numbers on account of the increase in the length of the course.

In the appended table is given the registration by years since the College opened:

TOTAL REGISTRATION OF UNDERGRADUATES BY YEARS IN THE NEW YORK STATE VETERINARY COLLEGE

1896–7	11	1906–7	86
1897–8	17	1907–8	80
1898–9	23	1908–9	93
1899–1900	30	1909–10	101
1900–1	45	1910–11	106
1901–2	51	1911–12	110
1902–3	62	1912–13	120
1903–4	86	1913–14	132
1904–5	108	1914–15	124
1905–6	88	1915–16	160

INSTRUCTION

With the multiplication of subjects that are constantly being brought into use by the veterinarian, the problem of arranging and carrying out a wise curriculum is becoming more and more difficult. Instruction in the basic sciences of anatomy, chemistry and physiology and the principles of pathology, medicine and surgery is no longer adequate to qualify men for practice or sanitary service. Veterinarians who are to meet the demands of the live stock owners must be proficient not only in the basic sciences but also in methods of diagnosis, the relation of nutrition to disease, prophylaxis, immunity and a multitude of phases in the methods for the control of epizoötics and the chronic infections. The teaching of these subjects in such a way as to insure on the part of the student an accurate knowledge and workable understanding of all the topics necessary for his efficiency places a heavy responsibility upon those giving the instruction.

In previous reports the method of departmental instruction that is followed has been explained. Each department is solving for itself the problems of instruction peculiar to the subjects it is teaching. We have been fortunate in being able to retain experienced teachers and to give recitation and laboratory instruction in small sections, which adds wonderfully to the efficiency of the teaching.

There is a standing committee to study the needs of the students and the requirements of the course from the professional point of

view. From its recommendations the faculty make from time to time such changes as seem necessary for the betterment of the course as a whole. The required three-year curriculum is appended. This is the last year when this will be given. Beginning with the fall of 1916 the freshman year of the four-year course was substituted for the first year of the three-year curriculum.

Prescribed Three-Year Course

First Year

	Number of course	Credit first term	Credit second term	Total actual hours
Inorganic chemistry	1	6	.....	135
Histology and embryology	6	3	5	215
Anatomy	1	3	.....	390
Anatomy	2	1	.....	
Anatomy	3	3	.....	
Anatomy	4	.....	5	
Physiology	10	3	.....	165
Physiology	12	.....	3	
Physiology	14	.....	2	
Feeding animals	1	.....	2	38
Animal breeding	2	.....	2	30
Total	.....	19	19	973

Second Year

	Number of course	Credit first term	Credit second term	Total actual hours
Anatomy	5	4	.....	300
Anatomy	6	2	.....	
Anatomy	7	.....	2	
Physiology	11	.....	1	15
Physiology	13	1	.....	15
Pharmacology	20	2	.....	30
Materia medica and pharmacy	21	2	.....	75
General pathology	40	5	.....	150
Parasitic pathology	44	1	.....	30
Animal parasites	22	2	.....	53
Small animal clinic	25	.....	1	45
Consulting clinic	53	.....	1	45
Bacteriology	43	.....	5	143
General surgery	30	.....	4	97
Horseshoeing and physical diagnosis	52	.....	3	45
Opthalmology	55	.....	1	15
Hygiene	56	.....	1	15
Total	.....	19	19	1,073

Third Year

	Number of course	Credit first term	Credit second term	Total actual hours
Urine analysis	15	1	.....	45
Diseases of small animals	22	.....	2	30
Materia medica and therapeutics	23	2	.....	30
Surgical exercises	31	1	.....	45
Special surgery	32	4	.....	60
Obstetrics	36	.....	4	60
Infectious diseases	42	.....	2	30
Special pathology	41	2	2	106
Small animal clinic	25	1	1	90
Consulting and medical clinic	53	1	1	90
Surgical clinic	34	1	2	180
Ambulatory clinic	37	1	1	90
Medicine	50	5	5	150
Total	.....	19	20	1,006

Each year there are several lectures and addresses given by distinguished non-resident veterinarians or others engaged in work of special interest to veterinary students. This year we were honored by the following persons, who either addressed the student body or spoke before the conference, to which the upper classmen were invited:

Dr. John Adams, professor of veterinary surgery, University of Pennsylvania, Philadelphia, Pa.

Dr. Adolph Eichhorn, chief of the Division of Animal Pathology, Bureau of Animal Industry, Washington, D. C.

Dr. J. G. Wills, chief veterinarian, Department of Agriculture, Albany, N. Y.

Professor H. E. Cook, dean of the School of Agriculture, St. Lawrence University, Canton, N. Y.

Dr. Harris Moak of the Certified Milk Commission, Brooklyn, N. Y.

Dr. Frank H. Miller, practitioner, New York City.

Dr. Cassius Way, chief of the veterinary inspection, Borden Milk Company, New York City.

Dr. Otto Faust, president of the New York State Veterinary Society, Poughkeepsie, N. Y.

Dr. W. G. Hollingworth, practitioner, Utica, N. Y.

In addition to the above, the following took an active part at the conference in a symposium on the Therapeutic Value of Biologic Products: Dr. N. S. Mayo, Abbott Laboratories, Chicago, Ill.; Dr. Walter E. King, Parke, Davis & Company, Detroit, Mich.; Dr. John Reichel, Mulford Company, Glenolden, Pa.; Dr. John F. DeVine, practitioner, Goshen, N. Y.; Dr. H. S. Beebe, practitioner, Albion N. Y.; Dr. Ira Buchanan, practitioner, Auburn, N. Y.

In the report for 1914–15 the changes that were believed to be advantageous to the teaching of clinical medicine and surgery were given in detail. The experience of the year has confirmed the wisdom of the rearrangement of the clinics. The ambulatory clinic, under the supervision of Dr. Udall, has been quite satisfactory. A very thorough system of examinations and note-taking has been put into operation in connection with the instruction

given in each case. During the nine months from October 1, 1915, to June 30, 1916, there were treated in this clinic 1,499 cases, of which 370 were horses, 684 cattle, 190 sheep, and 255 swine.

The consulting clinic, under the charge of Dr. Frost, has brought to the department of surgery a large number of cases valuable for teaching both major and minor surgery. There are from twenty-five to thirty-five animals constantly in the surgical hospital. This insures ample material for the teaching of clinical surgery. While the larger number of these cases were horses, a few other animals were brought to the surgical hospital for treatment.

The small animal clinic, under the charge of Dr. Milks, was well patronized, so that ample material for teaching purposes was constantly at hand. On several occasions the hospital was taxed to its capacity. The large amount of clinical material that is coming to this hospital has removed all doubt as to the ability to maintain clinical facilities for giving adequate instruction, from a thoroughly practical point of view, in the diseases of small animals.

In the appendix will be found a statement of the cases treated in each of the clinics. The advantages, especially for those who are preparing to practice veterinary medicine, of having a farming community immediately surrounding the College are very great. This is worthy of special emphasis because a large part of the work of veterinarians in the future will be in the country and with food-producing animals.

Number of Cases Treated in the Different Clinics by Years from 1907–8 to 1915–16, Inclusive, and the Number of Cases Treated from October 1, 1915, to June 30, 1916.

	Consulting and medical	Consulting	Surgical	Small animal	Ambulatory	Total
1907–8	175	....	48	103	125	451
1908–9	303	....	138	327	351	1,119
1909–10	332	....	141	274	695	1,442
1910–11	370	....	195	324	1,287	2,176
1911–12	215	....	245	351	937	1,748
1912–13	376	....	254	318	962	1,910
1913–14	348	....	212	492	917	1,969
1914–15	295	....	236	512	1,822	2,865
1915–16 (9 months)	....	451	175	397	1,499	2,522

The diagnosis work that is done at the College affords opportunity for senior and graduate students to acquire practical instruction and experience in methods of laboratory diagnosis. With proper instruction in this work and with a small equipment practitioners can make many of the positive diagnoses that are now obtained with much loss of time from laboratories. While many diagnoses require a fully equipped laboratory and experienced workers, veterinarians are becoming better prepared to make many of the determinations for themselves.

In an institution the functions of which are teaching and research, there is a modern tendency to emphasize the advantages for the so-called practical subjects and original research. With a developing subject like veterinary medicine the best possible research work may be concerned largely with improvement in the *presentation of subjects before classes.* The type of research mind needed is one that "keeps up to date in its correlations and brings the inspiration of the best and newest into each teaching day." The value of the institution will, in the last analysis, be determined by the thoroughness of its teaching, both in the basic sciences and in the practical subjects, quite as much as in specific research. The importance of research is unchallenged, but in building up an efficient veterinary service in the State teaching that will effectively impress the student with the knowledge

already available for better treatment of the sick and injured, as well as for the prevention and control of animal diseases, is just as serviceable and equally praiseworthy.

LABORATORY DIAGNOSIS

The diagnosis of morbid specimens for veterinarians of the State and the Commissioner of Agriculture has continued as usual. There has been a greater demand for diagnostic agents and anthrax vaccine than in previous years. The appended table gives a list of specimens and biologic products made during the nine months from October 1, 1915, to June 30, 1916.

EXAMINATIONS MADE AND THE AMOUNT OF TUBERCULIN, MALLEIN AND ANTHRAX VACCINE SENT OUT DURING THE NINE MONTHS ENDING JUNE 30, 1916.

Examinations for anthrax	79
Examinations for glanders (tissues)	9
Examinations for glanders (agglutination)	247
Examinations for poultry diseases	283
Examinations for rabies	108
Examinations for tumors	39
Examinations for tuberculosis	103
Examinations for black leg	7
Examinations for Johne's disease	4
Examinations, miscellaneous	160
Anthrax vaccine sent out (doses)	14,079
Mallein sent out (doses)	1,438
Tuberculin sent out (doses)	55,602
Retest tuberculin (doses)	25
Precipitated tuberculin (grams)	10

The diagnoses classified under miscellaneous include the following diseases, namely: chicken pox, contagious abortion, white scours, hepatitis, peritonitis, forage poisoning, hog cholera, necrobacillosis, parasites, including round worms, flat worms, mange, etc., pneumonia, meningitis, roup, intestinal calculi, bacillary white diarrhea, streptococcic mastitis, septicaemia hemorrhagica, chicken cholera, actinomycosis, blackhead, lymphadenitis, botryomycosis, calf pneumonia, contagious pleuro-pneumonia, coccidiosis, leukemia, arsenical poisoning, swine plague, scirrhous cord, hook worm disease, swamp fever, and some others.

Anthrax.—There were seventy-nine specimens received for examination for anthrax as compared with forty-seven last year. The number of positive cases was forty-four as compared with seventeen in 1914–15. Because of the persistence of this disease, it is recommended that veterinarians exercise the greatest possible care in the disinfection of infected stables and yards and in the disposal of carcasses. There is one locality from which we are receiving many specimens and where there seem to be many infected areas. It is very important that veterinarians recognize the serious economic significance of having farms permanently infected with anthrax. This means that for many years repeated vaccination will be imperative in that community if serious loss is prevented. The fact that the spores of anthrax remain in the soil for years requires for protection vaccination for a long time after the virus may seem to have disappeared. Anthrax was introduced into this country with hides from countries where the disease exists. It is well to be on guard for this affection in localities where in the past tanneries have existed or where they are now operating. Although the government requires the disinfection of hides imported into this country from infected territory, the resistance of anthrax spores to disinfectants is so great that it is possible for them to occasionally escape destruction and to be brought into a hitherto uninfected community.

In 1914–15 there were 11,270 doses of anthrax vaccine distributed, and in the nine months here reported there were 14,079 doses used. The appended table gives by months the number of specimens received for diagnosis and the results of the examinations.

SPECIMENS OF SUSPECTED ANTHRAX RECEIVED FOR EXAMINATION DURING THE NINE MONTHS FROM OCTOBER 1, 1915, TO JUNE 20, 1916

Month	Positive	Negative	Total
October	5	5	10
November	6	2	8
December	4	1	5
January	9	4	13
February	6	3	9
March	5	4	9
April	5	6	11
May	0	3	3
June	4	7	11
Total	44	35	79

ANTHRAX VACCINE SENT OUT DURING THE NINE MONTHS FROM OCTOBER 1, 1915, TO JUNE 30, 1916

Month	No. 1	No. 2	No. 1	No. 2	Total
October	500	500	0	0	1,000
November	500	500	50	0	1,050
December	0	0	0	0	0
January	1,000	1,000	0	0	2,000
February	0	0	0	0	0
March	0	0	200	0	200
April	1,240	0	64	0	1,304
May	3,700	3,700	480	430	8,310
June	0	0	115	100	215
Total	6,940	5,700	909	530	14,079

Glanders.—There were few specimens of tissues from cases of suspected glanders, and the number of samples of blood for the agglutination test was smaller this year than last. As we do not receive specimens from New York city, the reduction in the amount of this disease up State may be more apparent than real, because of the shorter time covered by this report. It is hoped that it will prove to be traced to the quarantine against glanders. It can be expected that if proper quarantine is enforced this disease will be minimized, if not entirely eliminated, from the country districts. The amount of mallein called for was greater

than last year. There has also been a small demand for precipitated mallein for the ophthalmic test. The appended tables give the details regarding the agglutinations that were made and the distribution of mallein to the State Department of Agriculture and to private practitioners.

AGGLUTINATION TEST FOR GLANDERS OCTOBER 1, 1915, TO JUNE 30, 1916

Month	1–500	1–800	1–1000	Negative	Total
October	14	2	8	26	50
November	17	1	10	45	73
December	10	2	2	24	38
January	8	0	0	3	11
February	0	0	0	5	5
March	2	0	0	1	3
April	0	0	0	2	2
May	16	1	3	19	39
June	6	0	2	18	26
Totals	73	6	25	143	247

NUMBER OF DOSES OF MALLEIN DISTRIBUTED IN THE STATE FOR THE NINE MONTHS FROM OCTOBER 1, 1915, TO JUNE 30, 1916, ARRANGED BY MONTHS

Month	State	Private	Total
October	200	0	200
November	10	10	20
December	500	1	501
January	0	5	5
February	0	0	0
March	200	0	200
April	200	5	205
May	5	102	107
June	200	0	200
Total	1,315	123	1,438

Rabies.—There has been a reduction from 165 specimens of suspected rabies last year to 108 for the nine months reported this year. The table will show that more than 50 per cent of these were negative. It is unfortunate that the attitude of the

people toward this disease is such that they will allow it to exist at all. If the lovers of dogs — and there are many — would see to it that suitable legislation relative to the licensing of dogs was enforced, the homeless dog, which is the greatest spreader of rabies, because most often attacked, would be eliminated, and, further, the large losses that dogs are causing the sheep industry would be prevented. The appended table gives the number of specimens received and the diagnosis.

TABLE GIVING BY MONTHS THE NUMBER OF SPECIMENS RECEIVED FOR THE DIAGNOSIS OF RABIES IN THE NINE MONTHS FROM OCTOBER 1, 1915, TO JUNE 30, 1916.

Month	Positive	Negative	Impossible	Total
October	13	5	1	19
November	7	9	0	16
December	5	5	1	11
January	5	7	0	12
February	8	4	0	12
March	3	5	1	9
April	3	7	0	10
May	1	9	1	11
June	3	5	0	8
Total	48	56	4	108

Tuberculosis.—There were a few more specimens of this disease received this year than last. Many of them came from animals that failed to react to the tuberculin test. In our last report I pointed out the limitations of tuberculin in diagnosing tuberculosis. It is very important for veterinarians to fully understand the conditions under which tuberculin fails to indicate the presence of the disease. Of the 31,665 doses of tuberculin sent to private practitioners 2,129 doses were returned. There is a slightly growing demand for precipitated tuberculin for the intradermal and conjunctival tests. The amount sent out, however, was only ten grams. There was also a call for a few doses of retest (double strength) tuberculin.

Specimens of Suspected Tuberculosis Received for Diagnosis from October 1, 1915, to June 30, 1916

Months	Positive	Negative	Total
October	1	4	5
November	5	8	13
December	7	14	21
January	7	9	16
February	4	5	9
March	6	9	15
April	1	6	7
May	3	8	11
June	4	2	6
Total	38	65	103

Tuberculin Sent Out for the Nine Months from October 1, 1915, to June 30, 1916

Date	State	Private	Returned	Total
October	1,700	3,761	10	5,451
November	2,985	4,574	74	7,485
December	5,125	3,721	653	8,193
January	1,500	3,532	1,220	3,812
February	3,500	2,405	0	5,905
March	3,000	3,514	41	6,473
April	3,505	4,639	22	8,122
May	2,550	3,212	85	5,677
June	3,000	2,507	21	5,486
Total	26,865	31,865	2,126	56,604

Distribution of Tuberculin for the Last Eight Years and from October 1, 1915, to June 30, 1916

Year	Number doses furnished State Department of Agriculture	Number doses sent to veterinarians in private practice	Total number of doses
1907–8	9,813	18,052	27,865
1908–9	15,302	17,587	32,889
1909–10	29,237	14,038	43,275
1910–11	29,781	17,648	47,429
1911–12	27,500	28,181	55,681
1912–13	27,265	34,107	61,372
1913–14	27,272	32,614	59,886
1914–15	16,910	37,137	54,047
1915–16	26,865	31,865	58,730

There is more tuberculin being used than heretofore. The amount furnished the Department of Agriculture has increased very much since July first.

Swine Diseases.— Hog cholera remains the most important infectious disease of swine in the State. A special number of the Cornell Veterinarian was devoted to hog cholera and the use of anti-hog cholera serum. A copy of this was sent to every licensed veterinarian in the State. Because of the importance of the disease and the assistance to be derived from the proper use of the serum this article prepared by Dr. Birch is reproduced in this report.

THE AMOUNT OF SERUM THAT HAS BEEN CALLED FOR BY VETERINARIANS AND SWINE OWNERS OF THE STATE AND DISTRIBUTED TO THEM DURING THE LAST FIVE YEARS AND THE NINE MONTHS FROM OCTOBER 1, 1915, TO JUNE 30, 1916

Year Ending	Quantity	Doses
September 30, 1911	43,340 c. c.	2,167
September 30, 1912	103,020 c. c.	5,151
September 30, 1913	157,340 c. c.	7,867
September 30, 1914	172,560 c. c.	8,628
September 30, 1915	244,760 c. c.	12,238
June 30, 1916	189,420 c. c.	9,471

In addition to the serum 2,531 c. c. of virus was sent to veterinarians for actively immunizing hogs against cholera.

Poultry Diseases.—There has been a steady increase in the number of dead and sick fowls received for examination. The treatment of poultry diseases is a very important part of veterinary practice that is largely neglected. The number of poultry in the State is estimated at 13,257,935, of which 12,655,664 are hens and chickens. In 1910 the value of the poultry in the State was placed at $7,879,388.

The loss from diseases caused by parasites and infections is estimated to be between 10 and 20 per cent. Some of the infections, such as white diarrhœa and tuberculosis, are well understood and can be controlled. It is hoped that more attention will be given to these diseases by practitioners and that funds may

soon be provided for special investigations concerning several of them. In the appendix will be found a valuable report by Dr. Pickens on leukemia in fowls.

The poultry industry is rapidly increasing in its economic significance, and the assistance that is possible for veterinarians to render in the prevention and control of the diseases of fowls is much greater than generally thought. It is also true that research work must be done on the nature of several of them before satisfactory methods for combating them can be formulated. To conserve this important part of animal industry, the diseases that annually more than decimate the investment must be brought under control.

Research

In a former report it was shown that in order to carry out at all satisfactorily the requirements of the statute establishing this College an experiment station where the work could be done to clear up, as far as possible, doubtful points in our knowledge of animal diseases and their treatment was necessary. In addition to that, fundamental research work is demanded in connection with several of the important infectious diseases. In obedience to these needs the University provided, free of charge, for the use of the college a farm for this purpose. A number of the more important experiments have been reported and others are in progress. It requires many years to satisfactorily complete many of these investigations. The researches which have progressed far enough to warrant the publication of the results at this time are as follows:

A study of the means by which hog cholera is disseminated by infected pork, and hog cholera and its prevention, by Dr. Birch; Leukæmia in fowls, and roup and chicken pox, by Dr. Pickens; Researches upon abortion of cattle, by Dr. Williams; A study of ovaritis in cattle, by Dr. Fitch; Further report of the diagnosis of open cases of tuberculosis, by Drs. Udall and Birch; A study of five members of the septicæmia hemorrhagica group of bacteria, by Mr. Besemer; The fermenting properties of *Bact. pullorum* and *Bact. sanguinarium,* by Dr. Goldberg; and The starch digesting properties of the saliva of the horse, by Dr. Hayden. These papers and reports are all fundamental in character.

The practical value of a knowledge of how hog cholera may be spread through infected pork is hard to estimate, for it enables at least one, and perhaps the most important, of its means of spreading to be eliminated. The greatest aid in checking the spread of an infectious disease is a knowledge of how its virus is disseminated. When the knowledge of these diseases is complete their control will be incidental because veterinarians and live stock owners will know what to do.

The report on abortion in cattle is of special interest. Although it leaves much to be attained, the findings are most valuable. The problem is a complex one, and its solution undoubtedly will not be accomplished until the findings of many researches and experiments are recorded. The results already obtained, however, will be very helpful to practitioners.

The economic significance of sterility has led to the study of the cause of ovaritis. This is a difficult problem closely associated with abortion. The work was undertaken to ascertain, if possible, to what extent sterility due to ovarian trouble is to be attributed to the bacillus of abortion and how much to other microorganisms. The report is not complete, but its importance warrants the publication of the preliminary results.

The study of the septicæmia hemorrhagica bacteria is of basic importance owing to the number of diseases in different species of animals caused by that group of bacteria. The question of significance is whether or not these bacteria are identical. The same question arises in regard to white diarrhœa of chickens and fowl typhoid. The important question is whether these diseases, now recognized as distinct maladies, are due to the same organism infecting young chicks on one hand and adult fowls on the other. For purposes of control it is essential that these technical points be carefully determined.

The very great economic importance of roup and chicken pox and the considerable losses from leukemia among fowls render the reports on these subjects of timely interest.

The report of the researches on the diagnosis of open cases of tuberculosis is of very great importance. The most interesting points brought out in this investigation are those pertaining to the proper protection of healthy cattle against infected ones that may

emain in herds after the supposedly diseased individuals have been removed; the importance of proper hygiene; and the limitations of each of the methods of diagnosis of open cases that have been proposed. These are matters of great practical importance to the owners of dairy cattle.

There is a no more important subject than physiology for the veterinarians to understand. There are, however, many phases in the functions of the organs of different species that are not thoroughly understood. This knowledge is necessary to properly direct treatment in time of sickness. One of these phases, namely, the amyloclastic activities of saliva in the horse, has been taken up by Dr. Hayden. This investigation has extended over considerable time and its results are worthy of careful study.

As previously stated, equally important work was done in the study of methods of teaching and in devising ways and means for a better presentation of the subjects to students. In this connection the most striking work was done by Dr. Hopkins in the preparation of a series of charts made from careful dissections to illustrate the anatomy of the cow. These will be of much assistance to the student in studying the anatomy of this species. As a large part of the work of the veterinarian is in treating the diseases of cattle, it is of the highest importance that the anatomy of the bovine be emphasized more than heretofore.

Extension Work

Although this College does not have an appropriation for extension work, there seems to be no more appropriate heading under which to group a number of its activities. Its function does not cease with the instruction of undergraduate students. The sciences involved in veterinary medicine are constantly changing and advancing. The discoveries in connection with the control of infectious diseases are causing continuous changes in sanitary work. New discoveries are pointing to new methods of treatment, new operations in surgery and many other things that for efficiency the practitioners should know but which from the very necessities of the case it is practically impossible for them to ascertain promptly.

Veterinarians are constantly encountering new conditions relative to nutrition, poisoning of animals, infections uncommon in their localities, and occasionally morbid conditions that require the assistance of experts to diagnose or to treat. It is of much value to the live stock interests of the State as well as to the practitioners to be able to obtain prompt assistance in dealing with those conditions which for want of facilities they cannot successfully do for themselves.

The preparation of reliable biologic diagnostic agents is a valuable aid to veterinarians and live stock owners alike. The diagnosis laboratory has enabled prompt determinations of the nature of outbreaks of disease in time to check their further spread. In these ways technical knowledge is being employed for the solution of the sanitary and economic problems involved in the treatment and control of animal diseases. The various activities of the faculty in assisting those having trouble in combating disease may be grouped as follows:

Conference for Veterinarians.— In January we held a two-day conference for veterinarians at the College. About 20 per cent of the legal practitioners of the State were present. The advances that had been made in certain of the more important subjects in veterinary medicine were presented by the most competent men obtainable. A symposium on the use of vaccines, bacterins, serums, etc., in the treatment and control of animal diseases was most helpful. Dr. A. Eichhorn of the Bureau of Animal Industry presented a valuable paper on the subject, which was followed by manufacturers of biologic therapeutic agents and veterinarians who had employed them in their practice. Such discussions bring about a better understanding not only of the virtue but also of the fraud that may exist in the newly developed and highly advertised "new remedies." The subject of infectious abortion in cattle and the protection of calves against disease were also discussed. The success of the conferences is largely measured by the interest taken in the papers, demonstrations and clinics by the constantly increasing number of veterinarians who attend.

Short Courses in Horseshoeing.— Courses in practical horseshoeing of six weeks duration are offered to the horseshoers of the

State. This has been done since the establishment of the farriery. There has been considerable demand from horseshoers for a place where they could receive scientific and practical instruction in this subject. Likewise, horse owners have demanded a better farriery service. In order to meet demands and to make it possible for these important artisans to receive this instruction the courses were established.

Fair.— Like other State educational institutions, this College has been requested by the State Fair Commission to make an exhibit each fall at the State fair. It is not possible for the College to make exhibits other than this. It has assembled a considerable collection of material illustrating such phases of certain diseases as may be of interest to animal owners. In these exhibits it strives to emphasize the nature of the disease, the procedure in making a diagnosis and as far as possible methods for its prevention. Competent and experienced men are present to answer questions of those interested in the prevention and control of animal diseases. From the many inquiries regarding this exhibit it is believed that much assistance is rendered to live stock owners by bringing to their attention the requirements necessary for the prevention and control of disease.

Correspondence.— Not least among the agencies for assisting veterinarians and live stock owners is the correspondence carried on by the College. As time goes on the inquiries are more numerous. This has developed a quite extensive correspondence with the practitioners as well as the live stock owners themselves. This consumes time and to that extent it interrupts other work. It is believed, however, that it is of much importance.

Miscellaneous.— One of the means of aiding the State is by furnishing technical information to various organizations having to do with the control of animal diseases. It happens frequently that members of the faculty are requested to assist in conventions or to serve on commissions relating to live stock sanitation. Considerable time is consumed in attending such conferences and meetings in connection with the veterinary service of the State. This is all time-consuming work and for that reason should be mentioned among the ways by which the College renders service to the public.

NEEDS OF THE COLLEGE

The College buildings have not been completed. The south wing to the main building has not been provided for. This is needed very much to furnish space for the overcrowded library, an amphitheatre adequate to hold the larger classes for demonstration lectures and conferences of veterinarians, and fireproof vaults for records, storerooms, and administration offices. It is difficult to do the required work properly without the necessary buildings.

There is need of a building for the State diagnosis work, the preparation of diagnostic agents and vaccines, the laboratories for bacteriology, pathology, post-mortem and parasitology, and to furnish research laboratories for botany, toxicology and chemistry. The use of the same laboratories for teaching undergraduates, for research and the diagnostic work is most unfortunate. This work has outgrown its facilities. The only practical solution is the construction of a new building dedicated to these purposes.

There is a steadily growing demand for provision for special research in connection with infectious abortion in cattle, influenza or shipping fever in horses, and several important diseases of fowls. The losses sustained by these maladies are enormous, and there is no hope of permanent relief until more knowledge is acquired concerning their causes and methods for their prevention. These are difficult problems and their solution cannot be expected promptly. Like cancer and smallpox in man, which have received for decades continuous investigation by most competent scientists, and still hold the secret of their etiology, these destructive diseases of animals may not for many years yield to the inquiries of science. However, the time has come when concerted and persistent effort should be made to find a means for curtailing the heavy losses annually sustained because of these maladies.

The growth of the work in each department has rendered it physically impossible to have it satisfactorily done with the present force. The cost of adequately teaching men to become competent veterinarians is rapidly advancing. This cannot be changed for the present. The multiplying of subjects that must be recognized somewhere in the curriculum requires additional and experienced instructors and much material. It also calls for more money

for equipment. The problems for research in connection with routine practice are numerous and should be solved. The losses from disease are constantly presenting new difficulties, and with the increase in value of live stock they become in proportion economically more serious. The college should be enabled to do all that is possible to reduce these losses. To do this it must have men and material.

Recommendations

It is recommended that the trustees ask the Legislature for the following appropriations for the year 1917–18:

For regular maintenance	$78,000
For special investigation on infectious abortion and sterility in cattle	5,000
For the south wing to James Law Hall and equipment of same	100,000

Financial Statement

The University charges $100 tuition for students not living in New York State. This adds a few hundred dollars to the operating funds each year from which the University has accumulated a small emergency fund for the use of this college. It often happens that the state appropriation is inadequate to meet certain demands. In such cases the trustees draw upon the tuition.

A fee is charged each student to cover material used by him in the laboratories and surgical exercises.

The clinics charge a fee from the owner of the patients to cover the expense of boarding the patient, transportation and medicines. A nominal charge to cover the cost of material is made for the tuberculin and mallein sent to private parties. The college supplies these substances free of charge to the Commissioner of Agriculture. A charge to cover cost of manufacture is made for the anti-hog cholera serum. The money derived from the laboratory fees, serum and clinic charges is returned as a revolving fund to the respective departments.

It is important to distinguish between material required by the teacher for teaching purposes and that used by the student in doing the work for himself. This necessitates the purchase of the same

kind of material from the appropriation for the teacher and from the revolving fund for the student's work. The balance shown in the statement of the revolving fund is due to the fact that the statement is made for July 1 instead of October 1. This balance belongs to the different departments and was largely used during the summer in putting in supplies. During the past years it has been possible to adjust the revolving fund so that staple articles necessary in the laboratory work can be purchased in advance. It is absolutely necessary for teaching purposes to do this. The statements of the revolving fund and of the maintenance are appended.

REPORT OF CIRCULATING FUNDS OF NEW YORK STATE VETERINARY COLLEGE AT CORNELL UNIVERSITY, OCTOBER 1, 1915, TO JUNE 30, 1916

Balance October 1, 1915		$3,984 65
Receipts October 1, 1915, to June 30, 1916:		
Tuition	$1,687 50	
Laboratory fees	3,668 44	
Clinics and medicine	3,552 90	
Tuberculin and mallein	1,424 60	
Hog cholera serum	3,073 92	
Horseshoeing	1,006 44	
Miscellaneous	329 11	
		14,742 91
		$18,727 56
Disbursements October 1, 1915, to June 30, 1916:		
Laboratory supplies	$1,848 59	
Farriery supplies	559 11	
Clinics and medicine	2,752 15	
Tuberculin and mallein materials	626 21	
Serum supplies	1,013 51	
Research work	402 59	
Extension work	717 59	
Contingencies	150 64	
		$8,070 39
Balance June 30, 1916		10,657 17
		$18,727 56

Expenditure of the Maintenance Fund for the New York State Veterinary College for the Year 1915–1916

(*Chapter* 725, *Laws of* 1915)

Personal service	$53,367 13
Fuel, light, power and water	2,260 95
Printing	613 88
Equipment	1,621 93
Supplies	6,289 69
Materials	1,159 51
Traveling expenses	136 75
Communication	780 63
General plant service	168 12
Contingencies	375 41
Total spent	$66,774 00
Balance lapsing to State	3,226 00
	$70,000 00

Expenditures for Equipment of Clinical Buildings for the College Year 1915–1916

(*Chapter* 531, *Laws of* 1914)

Balance unexpended October 1, 1915	$864 97
Spent October 1, 1915, to June 30, 1916	852 94
Balance lapsing to State	$12 03

As per itemized vouchers approved by the State Comptroller.

Respectfully submitted,

H. H. Haight,

Clerk.

In the appendix will be found details concerning the clinics and research work, which should be regarded as an integral part of this report.

It is a fair and just statement that the success of the College has been due to the co-operation of the faculty, to the end that the College may meet its obligations to the students and to the State.

V. A. MOORE,
Dean New York State Veterinary College
at Cornell University, Ithaca, N. Y.

APPENDIX

Containing Reports of Clinics and Research Work

REPORT OF THE AMBULATORY CLINIC FROM OCTOBER 1, 1915, TO JUNE 30, 1916 (NINE MONTHS).

D. H. Udall, F. F. Koenig, J. N. Frost, J. F. Shigley

The report of the ambulatory clinic for 1915–16 covers a period of only nine months. The relative number of cases for the same period last year is about the same.

Thirty-four students took work in the ambulatory clinic. They are required to write a report on each case, the symptoms and treatment of which must be presented in a systematic and detailed order. The securing of these notes entails much time and work. Some of our students seem mentally incapable of comprehending a uniform system for description. The difficulty of obtaining a concise, accurate description of a case is almost incredible. This, it seems to me, is due in part to indifference to the finer details of our problems — to an impression that a superficial or approximate knowledge of veterinary science is sufficient for all practical purposes. With our present organization, however, we are able to exact much better notes than formerly, so that they are beginning to be of some value as a reference record.

Reference to the distribution of material emphasizes the increasing importance of ruminants and swine in veterinary medicine. The tabulated report fails to bring out the close relation between abortion, metritis, and retained placentæ in cattle. In the great majority of the cases observed in this report the presence of one is equivalent to the presence of the other two. In the treatment of infections of the female generative organs we feel that much progress has been made during the past four years. While a comparison of the reports from year to year does not reveal any distinct difference, our notes indicate a more prompt and complete recovery. Our material is too limited for a definite conclusion, but a complete report will be made when sufficient data has accumulated.

Too much emphasis cannot be placed on the value of the ambulatory clinic as a means of teaching students, as well as instructors. Our conception of many of the more common diseases is quite largely determined by our personal experience in this clinic.

Financially the clinic barely pays expenses.

The following is a list of animals treated, the species affected, and the results:

TABULATED REPORT OF THE AMBULATORY CLINIC, OCTOBER 1, 1915, TO JUNE 30, 1916 (NINE MONTHS)

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
1. INFECTIONS AND INTOXICATIONS									
Abortion	3	22	....	....	24	....	....	1	.....
Actinomycosis	....	2	....	....	2	....	....	....	.....
Contagious pleuro pneumonia	1	....	....	....	....	....	....	1	.....
Hemorrhagic septicemia	....	1	....	....	....	....	....	1	.....
Hog-cholera	....	....	....	182	....	....	....	105	.....
Infectious keratitis	....	2	....	....	2	....	....	....	.....
Influenza	29	....	....	....	29	....	....	....	.....
Malignant head catarrh	....	1	....	....	....	....	....	....	.....
Necrobacillosis	....	1	....	....	....	....	....	1	.....
White scours	....	2	....	....	2	....	....	....	.....
2. DISEASES OF THE CIRCULATORY SYSTEM									
Azoturia	12	....	....	....	12	....	....	....	.....
Endocarditis, puerperal	....	1	....	....	....	....	....	1	.....
Heart weakness	3	....	....	....	1	....	....	2	.....
Pericarditis, traumatic	....	1	....	....	....	....	....	1	.....
Pericarditis, parasitic	1	....	....	....	....	....	....	1	.....
3. DISEASES OF THE RESPIRATORY SYSTEM									
Bronchitis	3	8	....	....	11	....	....	....	.....
Heaves	3	....	....	....	....	....	....	....	3
Hydrothorax, parasitic	1	....	....	....	....	....	....	1	.....
Hemorrhage, guttural pouch	1	....	....	....	....	....	....	1	.....
Laryngitis	3	....	....	....	3	....	....	....	.....
Nasal catarrh	1	1	....	....	2	....	....	....	.....
Pleuritis, traumatic abscess	....	1	....	....	....	....	....	1	.....
Pneumonia, aspiration	1	1	....	....	1	....	....	1	.....
Pneumonia, calf, catarrhal	....	2	....	....	....	2	....	....	.....
Pneumonia, croupous	2	....	....	....	....	....	....	....	.....
Pneumonia, traumatic	....	1	....	....	....	....	....	1	.....
4. DISEASES OF THE DIGESTIVE SYSTEM									
Ascariasis	2	....	1	6	6	2	....	1	.....
Ascites	....	....	1	....	....	....	....	1	.....
Choke	2	2	....	....	4	....	....	....	.....
Colic	29	4	....	....	32	....	....	1	.....
Constipation	....	....	....	2	1	....	....	....	1
Diarrhea	4	5	1	....	10	....	....	....	.....
Enteritis, hemorrhagic	....	4	....	....	2	....	....	2	.....
Esophagitis, traumatic	....	1	....	....	1	....	....	....	.....
Gastritis, traumatic	....	6	....	....	4	....	....	2	.....
Gastric ulcer	....	1	....	....	....	....	....	1	.....
Gastric catarrh	1	....	....	....	1	....	....	....	.....
Gastroenteritis	3	6	....	....	6	....	....	3	.....
Gastro-intestinal catarrh	3	1	....	....	4	....	....	....	.....
Hemorrhoids	....	....	....	2	....	....	....	2	.....
Impaction, colon	7	....	....	....	7	....	....	....	.....
Indigestion	16	35	1	....	49	....	....	2	.....
Intussusception	....	1	....	....	1	....	....	....	.....
Perverted appetite	....	....	....	30	30	....	....	....	.....
Prolapsus recti	....	1	....	1	1	....	....	1	.....
Rupture, colon	1	....	....	....	....	....	....	1	.....
Rupture, stomach	2	....	....	....	....	....	....	2	.....
Salivation	....	1	....	....	1	....	....	....	.....
Torsion, small intestine	3	....	....	....	....	....	....	3	.....
Tympany, acute	....	3	....	....	2	....	....	1	.....
Teeth, long	4	....	....	....	....	4	....	....	.....
Teeth, sharp	3	1	....	....	....	4	....	....	.....
Teeth, broken	....	1	....	....	....	1	....	....	.....
Teeth, worn	3	....	....	....	....	3	....	....	.....

TABULATED REPORT OF AMBULATORY CLINIC —(*Continued*)

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
5. DISEASES OF THE LIVER									
Icterus neonatorium	....	1	....	....	....	....	....	1	.....
6. DISEASES OF THE GENITO-URINARY SYSTEM, INCLUDING THE UDDER									
Agalactia	....	4	....	....	2	....	2	....	.....
Congestion, udder	....	2	....	....	2	....	....	....	.....
Contusion, teat	....	1	....	....	1	....	....	....	.....
Cyst, vaginal	....	2	....	....	2	....	....	....	.....
Dystocia	4	22	1	....	26	....	....	1	.....
Edema, udder	....	3	....	....	3	....	....	....	.....
Freemartin	....	1	....	....	....	....	....	....	.....
Hematuria	....	5	....	....	4	....	....	1	.....
Mastitis	....	25	....	....	8	10	1	2	4
Metritis	....	29	....	....	22	....	2	5	.....
Nephritis, toxic	....	....	140	....	....	....	....	25	.....
Omphalitis	1	....	....	....	1	....	....	....	.....
Paralysis, postpartum	....	1	....	....	1	....	....	....	.....
Paraplegia, postpartum, luxation of hip	....	5	....	....	4	....	....	1	.....
Paraphimosis	1	....	....	....	1	....	....	....	.....
Placenta, retained	....	56	....	....	56	....	....	....	.....
Prolapsus, uterine	....	5	....	....	3	....	....	2	.....
Prolapsus, vaginal	....	4	....	....	4	....	....	....	.....
Sterility	....	43	....	....	19	....	5	....	18
Stricture, teat	....	18	....	....	15	....	3	....	.....
Torsion, uterus	....	1	....	....	1	....	....	....	.....
Tumor, udder	....	2	....	....	2	....	....	....	.....
Vaginitis	....	7	....	....	7	....	....	....	.....
Vesicles, udder	....	1	....	....	....	1	....	....	.....
Wound, udder	....	2	....	....	1	1	....	....	.....
Wound, penis	....	1	....	....	1	....	....	....	.....
7. DISEASES OF THE SKIN									
Burns	2	....	....	....	2	....	....	....	.....
Dermatitis	1	3	....	....	4	....	....	....	.....
Edema	1	....	....	....	1	....	....	....	.....
Eczema	2	1	....	....	3	....	....	....	.....
Furunculosis	1	....	....	....	1	....	....	....	.....
Necrosis	....	1	....	....	1	....	....	....	.....
Ringworm	....	2	....	....	2	....	....	....	.....
Scratches	4	1	....	....	5	....	....	....	.....
8. DISEASES OF THE NERVOUS SYSTEM									
Encephalitis	....	1	....	....	....	....	....	1	.....
Epilepsy	2	....	....	1	....	....	2	1	.....
Myelitis	1	....	....	....	....	....	....	1	.....
Meningitis, pyemic	....	....	5	....	2	....	....	3	.....
Paraplegia	1	....	....	....	....	....	....	1	.....
Parturient paresis	....	7	....	....	7	....	....	....	.....
Tremors	....	3	....	....	3	....	....	....	.....
9. EXTERNAL DISEASES									
Abrasion	2	....	....	....	2	....	....	....	.....
Abscess	12	10	....	2	24	....	....	....	.....
Amputation, tails	....	....	10	....	....	....	....	....	.....
Arthritis	....	1	....	....	....	....	....	....	1
Atrophy, scapula reg	1	....	....	....	1	....	....	....	.....
Bursitis	6	....	....	....	1	....	....	....	2
Castration	26	3	10	18	57	....	....	....	.....
Contusion	4	....	....	....	4	....	....	....	.....
Corns	2	....	....	....	2	....	....	....	.....
Empyema, frontal sinus	1	2	....	....	2	....	1	....	.....
Footrot	....	5	....	....	5	....	....	....	.....
Fracture, hind leg	....	....	....	1	....	....	....	1	.....
Fracture, first phalanx	1	....	....	....	....	....	....	1	.....
Fracture, metatarsus	1	....	....	....	....	....	....	1	.....

TABULATED REPORT OF AMBULATORY CLINIC —(*Concluded*)

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Fracture, ilium	2	....	....	....	2	....	....	....	.....
Fracture, ribs	....	1	....	....	1	....	....	....	.....
Fracture, humerus	1	....	....	....	....	....	....	1	.....
Fracture, spine	1	....	....	....	....	....	....	1	.....
Fracture, pelvis	1	....	....	....	1	....	....	....	.....
Hematoma	2	....	....	....	2	....	....	....	.....
Hemorrhage, after dehorning	....	1	....	....	1	....	....	....	.....
Hernia	1	1	....	....	....	....	....	....	2
Keratitis	1	....	....	....	1	....	....	....	.....
Laminitis	1	....	....	....	....	1	....	....	.....
Luxation, stifle	....	1	....	....	....	....	....	1	.....
Lymphangitis	7	....	....	....	6	....	....	1	.....
Nail puncture	2	....	....	....	2	....	....	....	.....
Open joint	2	....	....	....	....	....	....	1	1
Ophthalmia, periodic	8	....	....	....	....	3	2	....	3
Papilloma, lid	1	....	....	....	1	....	....	....	.....
Phlegmon, limbs	3	....	....	....	3	....	....	....	.....
Pododermatitis	3	1	....	....	4	....	....	....	.....
Poll evil	1	....	....	....	....	1	....	....	.....
Ringbone	2	....	....	....	....	....	....	....	2
Sessamoiditis	1	....	....	....	....	....	1	....	.....
Shoe boil	1	....	....	....	....	....	....	....	1
Shoulder lameness	3	....	....	....	....	2	....	....	1
Shoulder galls	2	....	....	....	2	....	....	....	.....
Spavin	2	1	....	....	....	....	....	....	3
Sprain, tarsus	1	....	....	....	....	....	....	1	.....
Side bone	1	....	....	....	1	....	....	....	.....
Stiffness	1	....	....	....	1	....	....	....	.....
Strain, stifle muscles	2	....	....	....	2	....	....	....	.....
Teeth, sharp	3	1	....	....	4	....	....	....	.....
Teeth, long	2	....	....	....	2	....	....	....	.....
Teeth, worn	3	....	....	....	....	....	....	....	3
Teeth, wolf	2	....	....	....	....	....	....	....	2
Tendinitis	2	....	....	....	....	....	....	....	2
Tendovaginitis	1	....	....	....	....	....	....	....	1
Tumor, melanoma	1	....	....	....	....	....	....	....	1
Wounds	44	5	....	....	49	....	....	....	.....
10. POISONING									
Tobacco dip	....	1	....	....	....	1	....	....	.....
Lead arsenate	....	2	....	....	1	....	....	1	.....
Mouldy feed	1	....	....	....	1	....	....	....	.....
11. MISCELLANEOUS									
Chilling	1	....	....	....	1	....	....	....	.....
Debility, age, starvation, cast, etc	5	4	....	....	....	....	....	8	1
Dehorning	....	68	....	....	68	....	....	....	.....
Examination, health	1	3	....	1	....	....	....	....	.....
Examination, pregnancy	27	24	....	....	....	....	....	....	.....
Interruption of pregnancy	....	4	....	....	....	....	....	....	.....
Sputum examination	....	23	....	....	....	....	....	....	.....
Tuberculin test	....	142	....	....	....	....	....	....	.....
Totals	370	683	170	246	....	....	....	....	.....
Total cases									1,469

REPORT OF THE SURGICAL CLINIC FOR LARGE ANIMALS FROM OCTOBER 1, 1915, TO JULY 1, 1916

J. N. FROST and J. F. SHIGLEY

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Abscess	....	4	....	1	5	....	....	....	.....
Alveolar pericstitis	6	....	....	....	5	....	....	....	1
Arthritis	12	....	....	....	9	1	1	....	1
Bursitis	....	2	....	....	2	....	....	....	.....
Canker	1	1	....	....	....	1	....	1	.....
Castration	5	1	....	20	26	....	....	....	.....
Caudal myotomy	1	....	....	....	1	....	....	....	.....
Contracted tendons	2	....	....	....	....	2	....	....	.....
Contusions	1	....	....	1	2	....	....	....	.....
Corn, suppurating	1	....	....	....	1	....	....	....	.....
Cryptorchid	2	....	....	....	2	....	....	....	.....
Cystitis	1	....	....	....	1	....	....	....	.....
Degeneration of testicle	1	....	....	....	1	....	....	....	.....
Dehorning	....	3	....	....	3	....	....	....	.....
Diseased teeth	7	....	....	....	6	....	....	....	1
Entropium	1	....	....	....	1	....	....	....	.....
Fibroma	4	1	....	....	5	....	....	....	.....
Fistulous withers	16	....	....	....	14	....	....	2	.....
Foot rot	....	2	....	....	2	....	....	....	.....
Foreign body, shoulder	1	....	....	....	1	....	....	....	.....
Fracture, maxillary bone	1	....	....	....	1	....	....	....	.....
Fracture, occipital crest	1	....	....	....	1	....	....	....	.....
Gonitis	2	....	....	....	....	2	....	....	.....
Hernia, scrotal	....	....	....	20	20	....	....	....	.....
Hernia, umbilical	2	....	....	....	2	....	....	....	.....
Horn tumor	1	....	....	....	1	....	....	....	.....
Hygroma	....	2	....	....	2	....	....	....	.....
Intrissusception	....	2	....	....	2	....	....	....	.....
Joint, open	2	....	....	....	1	....	....	1	.....
Keratitis	1	....	....	....	1	....	....	....	.....
Lymphangitis	3	....	....	....	3	....	....	....	.....
Laminitis, acute	2	....	....	....	2	....	....	....	.....
Metritis	....	7	....	....	6	....	....	1	.....
Navicular bursa, open	1	....	....	....	1	....	....	....	.....
Nymphomania	4	....	....	....	4	....	....	....	.....
Odontome	2	....	....	....	2	....	....	....	.....
Open joint	2	....	....	....	1	....	....	1	.....
Open navicular bursa	1	....	....	....	1	....	....	....	.....
Open tendon sheath	1	....	....	....	1	....	....	....	.....
Parturient paresis	....	1	....	....	1	....	....	....	.....
Placenta retained	....	2	....	....	2	....	....	....	.....
Poll evil	1	....	....	....	1	....	....	....	.....
Purpura hemorrhagica	1	....	....	....	....	....	....	1	.....
Retained placenta	....	2	....	....	2	....	....	....	.....
Ringbones	6	....	....	....	4	1	....	....	1
Roaring	13	....	....	....	10	1	....	....	2
Scirrhous cord	1	....	....	....	1	....	....	....	.....
Scrotal hernia	....	....	....	20	20	....	....	....	.....
Sessamoiditis	2	....	....	....	....	2	....	....	.....
Shoe boil	1	....	....	....	1	....	....	....	.....
Shoulder tumor	6	....	....	....	6	....	....	....	.....
Spaying	4	....	....	....	4	....	....	....	.....
Spavin	4	....	....	....	3	1	....	....	.....
Sterility	....	4	....	....	3	....	....	....	1
Teeth, disease of	7	....	....	....	7	....	....	....	.....
Teeth, extraction of	2	....	....	....	2	....	....	....	.....
Tendons, contracted	2	....	....	....	....	2	....	....	.....
Tendon, sheath open	1	....	....	....	1	....	....	....	.....
Tendo-vaginitis	2	....	....	....	2	....	....	....	.....
Testicle, degeneration	1	....	....	....	1	....	....	....	.....
Thoroughpin	1	....	....	....	1	....	....	....	.....
Tumor, benign	5	1	....	....	6	....	....	....	.....

REPORT OF SURGICAL CLINIC — (*Concluded*)

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Tumor, malignant	3	....	....	....	1	....	....	2	.....
Tumor, melanotic	2	....	....	....	1	....	....	1	.....
Tumor, shoulder	6	....	....	....	6	....	....	....	.....
Umbilical hernia	2	....	....	....	2	....	....	....	.....
Withers, fistula of	16	....	....	....	14	....	....	2	.....
Wounds	15	1	1	....	17	....	....	....	.....
Total	192	36	1	62	258	13	1	12	7
Gross total									291
Counted twice									116
Net total									175

REPORT OF THE CONSULTING CLINIC FROM OCTOBER 1, 1915, TO JULY 1, 1916

J. N. Frost and J. F. Shigley

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Abscess	2	2	....	1	5	....	....	....	.....
Anal fistula	1	....	....	....	....	....	1	....	.....
Anchylosis of lumbar vertebrae	1	....	....	....	....	....	1	....	.....
Arthritis	14	....	....	....	10	3	1	....	.....
Azoturia	1	....	....	....	1	....	....	....	.....
Bronchitis	3	....	....	....	3	....	....	....	.....
Burns	2	....	....	....	2	....	....	....	.....
Carpitis	1	....	....	....	....	1	....	....	.....
Castration	13	....	....	19	32	....	....	....	.....
Caudal myectomy	1	....	....	....	1	....	....	....	.....
Colic	2	....	....	....	2	....	....	....	.....
Contracted heels	4	....	....	....	....	4	....	....	.....
Corns	5	....	....	....	5	....	....	....	.....
Cryptorchid	....	....	....	1	1	....	....	....	.....
Cyst, retention	2	....	....	....	2	....	....	....	.....
Dermatitis	11	....	....	....	11	....	....	....	.....
Diarrhea	2	....	....	....	....	2	....	....	.....
Eczema	7	....	....	....	7	....	....	....	.....
Edema	3	....	....	....	3	....	....	....	.....
Emaciation	2	....	....	....	....	....	....	2	.....
Examination for pregnancy	2	2	....	....	....	....	....	....	.....
Examination for soundness	19	1	....	....	....	....	....	....	.....
Exostosis	2	1	....	....	....	3	....	....	.....
Fistula, anal	1	....	....	....	....	....	1	....	.....
Fistula, withers	2	....	....	....	2	....	....	....	.....
Flatulence	2	....	....	....	2	....	....	....	.....
Fracture, mandible	2	....	....	....	2	....	....	....	.....
Gonitis	3	....	....	....	....	2	1	....	.....
Hair root infection	2	....	....	....	2	....	....	....	.....
Heaves	6	....	....	....	....	6	....	....	.....
Heels, contracted	4	....	....	....	....	4	....	....	.....
Hematome	1	1	....	....	2	....	....	....	.....
Hemaphrodite	....	1	....	....	....	....	....	1	.....
Hernia, scrotal	....	....	....	18	18	....	....	....	.....
Hernia, umbilical	....	....	1	....	1	....	....	....	.....
Hoof, deformed	....	3	....	....	....	3	....	....	.....
Imperforate anus	....	....	....	1	....	....	....	1	.....
Indigestion	8	2	....	....	10	....	....	....	.....
Infection, hair root	2	....	....	....	2	....	....	....	.....
Infection, umbilical	....	1	1	....	1	....	....	1	.....
Influenza	1	....	....	....	1	....	....	....	.....
Iritis, exudative	1	....	....	....	1	....	....	....	.....
Keratitis	12	1	....	....	10	....	1	....	2
Laminitis	3	....	....	....	3	....	....	....	.....
Leucorrhea	3	....	....	....	2	....	....	....	1
Lymphangitis	5	....	....	....	5	....	....	....	.....
Mammitis	....	3	....	....	3	....	....	....	.....
Mange	3	....	....	....	2	....	....	1	.....
Melanoma	1	....	....	....	1	....	....	....	.....
Metritis	3	1	....	....	3	....	....	....	1
Myectomy, caudal	1	....	....	....	1	....	....	....	.....
Navicular disease	4	....	....	....	....	4	....	....	.....
Necrosis, skin	3	....	....	....	3	....	....	....	.....
Opthalmia, periodic	12	....	....	....	7	....	2	....	3
Os pedis, sinking of	2	....	....	....	....	....	2	....	.....
Parasites, intestine	23	....	....	....	23	....	....	....	.....
Periproctitis	1	....	....	....	....	....	....	1	.....
Pink eye	2	....	....	....	2	....	....	....	.....
Pododermatitis	7	....	....	....	7	....	....	....	.....
Post mortems	7	4	3	3	....	....	....	....	.....
Pyemia	1	....	1	....	....	....	....	2	.....
Quarter crack	2	....	....	....	2	....	....	....	.....
Ringbone	3	....	....	....	2	....	1	....	.....
Roaring	1	....	....	....	1	....	....	....	.....
Scirrhous cord	1	....	....	....	1	....	....	....	.....
Scratches	11	....	....	....	11	....	....	....	.....

REPORT OF CONSULTING CLINIC — (*Concluded*)

NAME OF THE DISEASE	Horses	Cattle	Sheep	Swine	Recovered	Improved	Not improved	Died or killed	Undetermined
Scrotal hernia	....	....	....	18	18	....	....	....	.....
Sesamoiditis	2	....	....	....	....	2	....	....	.....
Sheath cleaned	9	....	....	....	....	....	....	....	.....
Shoe boil	2	....	....	....	2	....	....	....	.....
Shoulder tumor	12	....	....	....	12	....	....	....	.....
Side bones	2	....	....	....	....	2	....	....	.....
Sit fast	2	....	....	....	2	....	....	....	.....
Spavin	9	....	....	....	7	....	1	....	1
Sterility	8	2	....	....	7	....	....	....	3
Stomatitis	2	....	....	....	2	....	....	....	.....
Synovitis, pyemic	2	....	....	....	2	....	....	....	.....
Teeth, disease of	2	....	....	....	2	....	....	....	.....
Teeth, examination of	61	....	....	....	61	....	....	....	.....
Teeth, extraction of	2	....	....	....	2	....	....	....	.....
Teeth, long	6	....	....	....	6	....	....	....	.....
Teeth, sharp	22	....	....	....	22	....	....	....	.....
Tendon, contraction of	3	....	....	....	1	2	....	....	.....
Tendo-vaginitis	4	....	....	....	4	....	....	....	.....
Thrush	5	....	....	....	5	....	....	....	.....
Trochanteric lameness	3	....	....	....	3	....	....	....	.....
Tumor, benign	2	1	....	....	3	....	....	....	.....
Tumor, malignant	2	1	....	....	2	....	....	1	.....
Tumor, melanotic	1	....	....	....	1	....	....	....	.....
Umbilical infection	....	1	1	....	1	....	....	1	.....
Urticaria	4	....	....	....	4	....	....	....	.....
Withers, fistula of	2	....	....	....	2	....	....	....	.....
Wounds	32	2	....	....	34	....	....	....	.....
Total	447	30	7	61	423	38	12	11	11
Gross total									545
Counted twice									94
Net total									451

The report for the surgical and consulting clinic covers a period of nine months from October 1, 1915, to July 1, 1916.

The number of cases treated in the surgical clinic would average the number treated in former years. Several of the cases were of an unusual and important class. The number of cattle treated has increased while the number of horses has decreased.

The cases treated in the surgical clinic may all be classified as major surgery and the animals were kept in the hospital that the student might have an opportunity to learn and apply the after treatment, which is an important part in the education of a student. It also furnishes an opportunity to see the effect of the treatment applied and the results obtained.

The consulting clinic was transferred to the Department of Surgery October 1, 1915. This affords a great number of cases of minor surgery and better balanced teaching of surgery.

In reporting for the consulting clinic, attention is called to the increase of over 100 per cent in the number of cases treated and a greater proportion of cattle than in former years.

Intussusception of the Small Intestine of a Cow

The patient was a grade Guernsey heifer sent to the surgical clinic by the professor of the ambulatory clinic with a diagnosis of intussusception and with the following history:

The animal was found standing with hind feet stretched backward and treading continuously. Feces had been passed only once in past twenty-four hours, and animal had not eaten in forty-eight hours. Milk secretion had stopped. Pulse 100, temperature 101.4; breathing was rapid and shallow. Mucous membranes were pale, extremities were cold and the animal was shivering. Peristalsis was fair on the left side but on the right side was suppressed and pressure on the lower part of abdomen on this side brought symptoms of pain. Rectal examination found the posterior intestinal tract empty except for bloody mucus. Intussusception of small intestines was found on right side of abdominal cavity.

Owing to the drifted condition of the roads the animal was not brought to clinic for forty-eight hours after the diagnosis was made.

March 20, 1916. When brought to clinic animal was very weak and the movements were stiff and unsteady. The abdominal

muscles were contracted and tense with the animal straining continually and passing small quantities of bloody mucus. Pulse 120, temperature 101.8. Respiration was rapid and shallow. Peristalsis and contraction of the rumen were absent. Operation: An area in the right flank was shaved, washed with gasoline and painted with tincture of iodine. The animal was given one ounce of chloral hydrate in two quarts of water per rectum and placed on the operating table. Cocaine was injected locally over the line of operation.

An incision was made through the skin, the muscle fibres were separated and the peritoneum punctured. An assistant then grasped the intestine through the rectum and brought the intussuscepted portion up through the incision.

The jaws of two pair of dressing forceps were covered with rubber tubing to lessen the injury to the intestine and one pair was clamped on each side and about two inches from the intussuscepted intestine. End to end anastamosis was then performed. The mesentery was cut away from the diseased portion of intestine, the blood vessels ligated and the intestine removed. The cut ends of the intestine were then sutured with two rows of intestinal sutures bringing the serous coats together. The cut mesentery was then stitched to this portion of the intestine. During the operation the intestine was frequently washed with warm normal salt solution and sterile cheese cloth was used for swabs. The skin and muscle wounds were closed with a single row of sutures.

The animal was then removed from the table and given an enema of warm salt solution and stimulating drench of capsicum and nux vomica. Drench and enema were repeated six hours later.

March 21, 1916. Eighteen hours after the operation the animal had passed feces three times, drunk a pail of warm water and eaten a quart of bran. Drench and enema repeated twice daily. Pulse 90, temperature 101.8.

March 22, 1916. Fair amount of feces mixed with mucus passed during the night. Ate bran and alfalfa and drank water. Drench and enema twice daily. Pulse 88, temperature 101.8.

March 23, 1916. Animal eating, drinking and chewing cud. Feces passed without mucus. Drench repeated but enema was discontinued. Pulse 72, temperature 101.6.

March 24, 1916. Improvement continues. Pulse 70, temperature 101.6.

March 25, 1916. Improvement continues. Pulse 68, temperature 101.8.

March 26, 1916. Feces passed with large amount of clear mucus. Pulse 65, temperature 101.7. During the day cow aborted a two months' fœtus. The uterus was irrigated with one-quarter per cent lugol's solution and the membranes were expelled. The skin wound showed slight suppuration and was painted with tincture of iodine.

March 27, 1916. Pulse 70, temperature 102.2. Slight discharge from vagina. External genitals were washed with one-half per cent wescol solution and vagina irrigated with normal salt.

March 28, 1916. Stitches removed from the wound. Slight suppuration. Painted wound with tincture of iodine. Pulse 65, temperature 101.

March 29, 1916. Small amount of necrotic tissue removed from the skin wound. Painted surface of wound with tincture of iodine. Irrigated the uterus with normal salt solution. Milk is now being secreted. Pulse 60, temperature 101.

March 30, 1916. Feces normal, animal on full diet, milk increased. Pulse 60, temperature 101.6. The external wound was treated with iodine daily until healing was complete.

REPORT OF THE CLINIC FOR SMALL ANIMALS, OCTOBER 1, 1915, TO JULY 1, 1916

H. J. MILKS and W. E. MULDOON

Department of Materia Medica and Small Animal Clinic

The clinic for small animals has been up to its previous standards both as to number and variety of cases treated. There has been sufficient material for instruction for the time alloted to the subject and frequently it has been necessary to devote considerable additional time to it.

Since most of the cases remain in the hospital, the students have an opportunity to become familiar with the different diseased conditions and to observe the effects of treatment upon them.

A great deal of importance has been given to the subject of anesthesia of these animals. All operations with the exception of a few very minor ones have been performed under anesthesia. Considerable time also has been devoted to surgical technic, in so far as this differs from that used upon the larger animals.

EXTERNAL DISEASES

Diseases of the Eye

	Dog	Cat	Total
Conjunctivitis	3	.....	
Cornea, wound of	1	.....	
Cornea, opacity of	1	.....	
Cataract	1	.....	
Eyeball, enucleation of	1	.....	
Keratitis	4	.....	
Orbital gland, removal of	1	.....	
	12	.....	
			12

Head and Neck

Abscess on neck	2	.....	
Carcinoma of mouth	1	.....	
Papilloma of mouth	1	.....	
Teeth, worn out	....	1	
Teeth, clipping (ferret)	....	1	
Goitre, cystic	1	.....	
Tumefied submaxillary glands	2	.....	
Otorrhea, catarrhal	5	.....	

	Dog	Cat	Total
Otorrhea, parasitic	3	5	
Wounds on head	1	.....	
Wounds on neck	....	1	
	16	8	
			24
Anterior Extremities			
Amputation of foot	....	1	
Cutting claws (bear)	1	1	
Cutting claws (ferret)	....	1	
Abscess of shoulder	....	1	
Carpus, fracture of	1	.....	
Injury to leg	1	.....	
Wound on leg	3	.....	
Wound on foot	2	.....	
Wound on shoulder	2	.....	
Lameness	1	.....	
	11	4	
			15
Thorax and Abdomen			
Castration	3	15	
Carcinoma of mammary gland	3	.....	
Oophorectomy	108	28	
Hernia, umbilical	1	.....	
Fistula of abdomen	2	.....	
Rectum, prolapse of	....	1	
Proctitis	1	.....	
	118	44	
			162
Posterior Extremities			
Femur, fracture of	2	2	
Metatarsus, fracture of	1	.....	
Paralysis	2	.....	
Contusions	2	.....	
Wound on hock	1	.....	
Wound on hip	1	.....	
Wound on foot	1	1	
	10	3	
			13
Vertebrae, Pelvis and Tail			
Amputation of tail	3	.....	
Wound on tail	1	.....	
	4	.....	
			4

Miscellaneous

	Dog	Cat	Total
Autopsy	4	3	
			7

INTERNAL DISEASES

Infectious Diseases

Distemper	30	.....	
Rabies	1	.....	
Infectious enteritis	....	1	
Tuberculosis	....	1	
	31	2	
			33

Respiratory System

Catarrh, nasal	1	.....	
Laryngitis	2	.....	
Bronchitis, parasitic	3	.....	
Pneumonia	1	.....	
Dyspnea	1	.....	
Cough, chronic	1	.....	
	9	.....	
			9

Digestive System

Gastritis	4	.....	
Gastric rupture	1	.....	
Stomatitis	....	1	
Enteritis	1	.....	
Hemorrhagic gastro-enteritis	1	.....	
Hepatitis	1	.....	
Indigestion	8	2	
Constipation	2	.....	
Worms, hook (uncinaria)	6	.....	
Worms, round	1	2	
Worms, tape	6	.....	
	31	5	
			36

Genito-Urinary System

Calculi, urethral	1	.....	
Mastitis	2	.....	
Inflammation of prepuce	1	.....	
Paraphymosis	1	.....	
Vagina, inflammation of	1	.....	
Metritis	1	.....	
Sterility	1	.....	
	8	.....	
			8

Diseases of the Skin	Dog	Cat	Total
Dermatitis	1	.....	
Urticaria	1	.....	
Mange, sarcoptic	1	8	
Mange, follicular	8	.....	
Eczema	15	.....	
Ringworm	4	.....	
Fleas	13	.....	
Lice	1	.....	
	44	8	
			52
Nervous System			
Epilepsy (?)	2	2	
Neuritis	2	1	
	4	3	
			7
Miscellaneous			
Rachitis	3	.....	
Rheumatism	1	.....	
Feather eating (parrot)	....	1	
Suspected rabies	1	.....	
Destroy	4	2	
Observation	7	1	
	16	4	
			2

SUMMARY

	Bear	Dogs	Parrot	Cats	Ferrets	Total
Diseases of the eye	....	12	.....	.....	.....	12
Head and neck	....	16	.....	7	1	24
Anterior extremities	1	10	.....	3	1	15
Thorax and abdomen	....	118	.....	44	.....	162
Posterior extremities	....	10	.....	3	.....	13
Vertebræ, pelvis and tail	....	4	.....	.....	.....	4
Miscellaneous	....	4	.....	3	.....	7
Infectious diseases	....	31	.....	2	.....	33
Respiratory system	....	9	.....	.....	.....	9
Digestive system	....	31	.....	5	.....	36
Genito-urinary	....	8	.....	.....	.....	8
Diseases of the skin	....	44	.....	8	.....	52
Nervous system	....	4	.....	3	.....	7
Miscellaneous	....	16	1	3	.....	20
	1	317	1	81	2	402

Total number of cases	402
Counted twice	5
Number of animals	397

REPORT OF THE DEPARTMENT OF OBSTETRICS AND OF RESEARCH IN THE DISEASES OF BREEDING CATTLE.

W. L. WILLIAMS

The instruction work in veterinary obstetrics is being gradually shifted in an effort to adjust the teaching to the needs of the State. The diseases of the genital organs of animals have been largely taught as a part of obstetrics since the foundation of the college. The live stock interests in the State of New York have shifted greatly in comparative value. The numbers of horses and cattle have remained almost static, while the average value has increased greatly. The relative demands for veterinary services for horses and for cattle have been reversed. Especially has this been true in connection with the diseases of breeding animals. Few horses are now bred in the State; most of those used are imported from the western states.

The demands for milk and other dairy products have increased enormously the importance of our dairy industry. Dairying is dependent wholly upon the reproductive functions. The increasing demands for dairy products have brought increased pressure upon dairy animals. In order to meet the demands for milk, the cows are more intensely bred, fed and housed. These three elements tend to reduce vitality and decrease the power of resistance to disease, especially chronic maladies like tuberculosis and the sterility-abortion group of diseases. These changes have brought veterinary obstetrics and the diseases of breeding cattle into extraordinary prominence and given to the sterility-abortion group and to tuberculosis the first place in economic and sanitary importance amongst all diseases of domestic animals. The diseases of the genital organs of cattle have come to be a very serious menace to the dairy industry of the State. The very existence of some herds is in peril because of disease. Thus in our report of research work in herd B it required an addition of 20 per cent annually by purchase, with all the heifer calves they could raise, in order to keep up the size of the herd. Again, in herd A, the group of eighteen heifers in first pregnancy in 1912, after four years in the dairy, counting the original eighteen, their

daughters, and granddaughters, the group has decreased in numbers seriously. These are not isolated examples, but a condition commonly observed.

These conditions render changes in veterinary instruction mandatory, and at no other point so insistent as in obstetrics and genital diseases of cattle. We recognized these changing conditions early in our teaching work, and have gradually and constantly shifted the emphasis from horses to cattle. Special efforts have been made since the establishment of our ambulatory clinic to secure and hold all available dairy practice. While the director of this clinic has been changed from time to time, the policy in dealing with the diseases of dairy cattle has been constant.

The clinical teaching in obstetrics is conducted chiefly by the ambulatory clinic. The class work is being devoted more and more each year to those questions affecting the dairy interests of the State. The students are recognizing more clearly each year that the dairies of the state offer the highest opportunity for scientific veterinary service, and the members of each succeeding class are showing a more intelligent interest in obstetrics and the diseases of the genital organs of cows, which so directly affect dairy efficiency.

HOG CHOLERA TRANSMISSION THROUGH INFECTED PORK

R. R. BIRCH
Veterinary Experiment Station

There is no other acute infectious disease of animals which is so widespread as hog cholera. It occurs in almost, if not quite, all countries in which swine are raised, and in some countries there are few large areas entirely free from it. While it is most prevalent near the more important shipping routes and in localities where large numbers of hogs are raised, it nevertheless appears frequently on remote farms and in localities far removed from busy traffic routes and centers. Its appearance in these seemingly well isolated places has been puzzling, for it is well known that it is caused by a specific virus, and that whenever it appears in a herd, the virus has in some manner been transferred to the herd from other infected animals.

Hog cholera virus, while it is not known to multiply outside the bodies of swine, is very tenacious and resists most natural destructive influences for long periods of time. A very small quantity* of it will infect an animal, and it is therefore commonly supposed that such casual carriers as crows, buzzards, and also various domestic animals not themselves susceptible to hog cholera, are in a large measure responsible for the many seemingly mysterious appearances of the disease. While the facts at hand do not admit doubt concerning the possibility of hog cholera virus transmission by these carriers there are good reasons to doubt whether they possess the degree of importance usually attributed to them. Circumstances seem to point to some important means of transmission less precarious than is furnished by such carriers.

Hogs that are fed garbage very frequently contract cholera and garbage often contains pork trimmings. Since garbage feeding is habitual both with farmers who feed only their own kitchen refuse and with men who make a business of removing and feeding city garbage, it seems reasonable to suppose that this practice

* King places the minimum fatal dose of hog cholera virus for a 50 lb. pig somewhere between 1/215 and 1/300 c.c. In his experiments the doses were administered intramuscularly.

may be responsible for many new herd infections. Further evidence supporting this belief is found in the facts that marketing the seemingly well animals in newly infected herds is a common practice, and that hog cholera virus appears in the blood stream of infected animals quite early in the course of the disease.

In the past, very little importance seems to have been attached to the transmission of the hog cholera through infected bits of pork. Dr. James Law mentions pork trimmings as a possible source of infection, but he lays special stress on dangers incident to feeding slaughter house refuse. Hutyra and Marek make no mention of market pork as a possible means of hog cholera transmission, and neither do Friedberger and Fröhner. Dr. M. Dorset in summarizing the various channels of inter-herd spread of the disease makes no mention of infected pork trimmings. So far as we know the first outbreak traced with any degree of accuracy to infected market pork was one in Canada which McGilvray reported in 1912. Even that outbreak seems to have been regarded as an exception for very little has been done looking toward the prevention of this means of hog cholera transmission.

Anti-hog cholera serum has removed one of the greatest obstacles in the way of hog cholera control. Not only does it protect herds in which the disease is just starting and prevent its appearance in other threatened herds, but it prevents, or should prevent, these herds from being shipped to market at times when they are in condition to infect other swine. It thus removes an almost unbearable hardship to swine breeders that otherwise would accompany the enforcement of strict sanitary measures to prevent shipping cholera infected hogs. It has given good reason to hope for the more complete control or eradication of hog cholera, and in so doing it has centered the efforts of a large number of veterinarians on a more thorough study of the disease itself, and on sanitary measures for its control. Since it cannot be effectively controlled as long as any one common means of transmission remains unknown or unheeded, it has seemed desirable to procure exact experimental data on the effects of feeding susceptible pigs bits of pork such as might be found in garbage.

The experiments have been conducted with three kinds of pork: kind were taken from carcasses that would have passed inspection, fresh, refrigerated, and cured. Some of the specimens of each

and others were taken from carcasses that would have been condemned. In all the experiments, before the specimens were removed for feeding, the hams were scalded and scraped as is done in butchering. Except as otherwise stated the material fed consisted of all or a part of the head of a femur together with adjacent parts. With one exception, experiment No. 1 in table No. 2, the hams all came from small shoats weighing less than one hundred pounds each, a fact which might have considerable influence on results obtained from feeding cured pork. Large hams would naturally be expected to harbor virus in their depths with somewhat greater regularity than small ones when both are subjected to killing influences that work from without.

The susceptible pigs to which the pork trimmings were fed were isolated with great care. In the earlier experiments small fly-tight pens were constructed of screen and matched lumber for this purpose. These were located on a hill several hundred feet from hog yards of any kind. When infection occurred in a pen it was immediately burned, and a new one was constructed on fresh soil for further experiments. The pigs fed in later experiments were placed in small individual fire brick pens so constructed that the attendant could not touch the pigs within. Food and water were introduced through a joint of tile. After each experiment the pen used was cleaned out and a wood fire was kindled inside and allowed to burn for several hours. Thus, in all cases, heat, rather than disinfectants, was used to destroy the virus. Most of the pigs were isolated a week or more before being fed and in no case did disease appear previous to feeding. In all cases the experimental pigs were selected from a herd of susceptible animals, and, except as noted, disease did not appear in the herd subsequent to the time the animals were removed. These two facts practically exclude the possibility that any of the experimental animals were infected prior to the time at which they were isolated.

In judging the part played by meat inspection in removing cholera infected carcasses from the market, the federal meat inspection regulations have been selected as a standard, because most of the meat inspected in the country is inspected by federal employees or by others who follow the federal regulations quite closely. Following are the paragraphs that govern ante-mortem and post-mortem inspection in their relation to hog cholera:

Regulation 9, section 2, paragraph 2. "All hogs plainly showing on ante-mortem inspection that they are affected with either hog cholera or swine plague shall be marked 'U. S. condemned' and disposed of in accordance with section 8 of this regulation.

Regulation 9, section 2, paragraph 3. "If a hog has a temperature of 106° F. or higher and is of a lot in which there are symptoms of either hog cholera or swine plague, in case of doubt as to the cause of the high temperature, after being marked for identification, it may be held for a reasonable time under the supervision of an inspector, for further observation and taking of temperature. Any hog so held shall be reinspected on the day it is slaughtered. If upon such reinspection, or, when not held for further observation and taking of temperature, then on the original inspection, the hog has a temperature of 106° F. or higher, it shall be condemned and disposed of in accordance with section 8 of this regulation.

Regulation 9, section 2, paragraph 6. "All animals which, on ante-mortem inspection, do not plainly show, but are suspected of being affected with, any disease or condition that, under these regulations, may cause condemnation, in whole or in part, on post-mortem inspection, shall be so marked as to retain their identity as suspects until final post-mortem inspection, when the carcasses shall be marked and disposed of as provided elsewhere in these regulations, or until disposed of in accordance with section 7 of this regulation.

Regulation 9, section 4, paragraph 1. "All hogs, even though not themselves marked as suspects, which are of lots one or more of which have been condemned or marked as suspects under section 2 of this regulation for either hog cholera or swine plague, shall, so far as possible be slaughtered separately and apart from all other animals passed on ante-mortem inspection."

Post-Mortem Inspection.

Regulation 11, section 4, paragraph 1. "The carcasses of all hogs marked as suspects on ante-mortem inspection shall be given careful post-mortem inspection, and if it appears that they are affected with either acute hog cholera or swine plague they shall be condemned.

Regulation 11, section 4, paragraph 2. "Carcasses of hogs which show acute and characteristic lesions of either hog cholera or

swine plague in any organ or tissue, other than the kidneys or lymph glands, shall be condemned. Inasmuch as lesions resembling lesions of hog cholera or swine plague occur in the kidneys and lymph glands of hogs not affected with hog cholera or swine plague, carcasses of hogs in the kidneys or lymph glands of which appear any lesions resembling lesions of hog cholera or swine plague shall be carefully further inspected for corroborative lesions. On such further inspection —

(a) "If the carcass shows such lesions in the kidneys, or in the lymph glands or both, accompanied by characteristic lesions in some other organ or tissue, then all lesions shall be regarded as those of hog cholera or swine plague, and the carcass shall be condemned.

(b) "If the carcass shows in any organ or tissue, other than the kidneys or lymph glands, lesions of either hog cholera or swine plague which are slight and limited in extent, it shall be passed for sterilization in accordance with regulation 15.

(c) "If the carcass shows no indication of either hog cholera or swine plague in any organ or tissue other than the kidneys or lymph glands, it shall be passed for food, unless some other provision of these regulations requires a different disposal."

Most of the virus used in the experiments was the same as was used in our routine work of serum production. It was of an exceedingly virulent strain obtained originally from Dr. W. B. Niles of Ames, Iowa. Pigs inoculated with 2 c. c. of this virus were usually ready to kill for virulent blood in seven days. In the remainder of the experiments the virus used was obtained from Dr. A. D. Fitzgerald, Columbus, Ohio. This also was of a highly virulent strain.

The method of securing carcasses that would pass inspection was to inject small shoats with 2 c. c. each of virulent blood and record temperatures every twenty-four hours subsequent to injections. When a decided elevation was recorded the pig was killed and autopsied. Then the ham was removed and scalded and a specimen secured for feeding. In each case the virus was injected into the right ham and the specimen fed was secured from the left ham. Complete data concerning these animals appear in table No. 1. Relative to the interpretation of results it should be stated

that, except as noted, all the lesions produced were of the acute form of hog cholera, and all the animals that sickened displayed symptoms similar to those produced by that disease. The term "typical lesions of cholera" as used in all the tables indicates that animals in reference revealed on autopsy petechiæ in the kidneys, and in addition characteristic hemorrhages (petechiæ and ecchymoses) in one or more other organs.

The animals that became infected were killed when severe symptoms developed in order that their blood might be used to hyperimmunize hogs in the routine of serum preparation.

TABLE No. 1

SHOWING TEMPERATURES, SYMPTOMS AND LESIONS OF PIGS FROM WHICH THE SPECIMENS FED WERE TAKEN

(Tables 2, Section b; 3, Section b; and 4, Section b.)

Number of pig	Date injected	Date killed	Temperature when killed	Symptoms noted	Lesions found*	Number of experiments in which specimen was fed
106[2]	Jan. 20	Jan. 23	106.2°	None	None	Experiment No. 6. Table No. 2, Sec. b.
107	Feb. 20	Feb. 23	105.4°	None	Mucosa of bladder**	Experiment No. 7. Table No. 2, Sec. b.
108	Mar. 30	April 2	105.6°	None	Slight inguinal lymph gland.** Right sublumbar lymph gland.** Left sublumbar and cardiac lymph glands.*	Experiment No. 8. Table No. 2, Sec. b.
109	Feb. 2	Feb. 6	105.6°	None	None	Experiment No. 9. Table No. 2, Sec. b.
110	April 15	April 18	Below 106.0°	None	None	Experiment No. 10 Table No. 2, Sec. b.
111	Oct. 21	Oct. 25	105.7°	None	None	Experiment No. 11. Table No. 2. Sec. b.
112	Jan. 26	Jan. 30	105.2°	None	None	Experiment No. 12. Table No. 2, Sec. b.
113	Jan. 26	Jan. 31	104.3°	None	None	ExperimentNo. 13. Table No. 2, Sec. b.
126	Jan. 22	Jan. 26	104.6°	None	None	Experiment No. 26. Table No. 3, Sec. b.
127	Jan. 22	Jan. 26	105.2°	Slight dullness.	Three or four mesenteric lymph glands**	Experiment No. 27. Table No. 3, Sec. b.
152	Nov. 20	Nov. 24	105.1°	None	Gastro-hepatic lymph glands*	Experiment No. 53. Table No. 4, Sec. b.
153	Nov. 20	Nov. 25	105.0°	None	None	Experiment No. 54. Table No. 4, Sec. b.

1. * Congestion. ** Slight hemorrhage. *** Marked hemorrhage.

TABLE No. 2

SHOWING RESULTS OF FEEDING FRESH PORK TO SUSCEPTIBLE PIGS

Section a. Pork from carcasses that would not pass inspection.

Number of experiments	Source of infected material	Quantity fed	Pig number	Date of feeding	Symptoms appeared	Date of death	Remarks
1.....	Rind and fat from shoulder.....	4 lbs.....	1 and 2	July 26–30	No symptoms	.....	Pigs later proved susceptible.
2.....	Flesh and bone.....	3 lbs.....	3 and 4	Oct. 9	Oct. 15	Oct. 20	Pigs killed when very weak. Typical lesions of cholera in both.
3.....	Flesh and bone.....	3 lbs.....	5 and 6	Oct. 20	Oct. 28	Nov. 3	Pigs killed when very weak. Typical lesions of cholera in both.
4.....	Flesh and bone.....	2 oz.....	7 and 8	Oct. 28	Nov. 8	Nov. 13	Pigs killed when very weak. Typical lesions of cholera in both.
5.....	Flesh and bone.....	1 oz.....	9 and 10	Jan. 7	Jan. 12	Jan. 15	Pigs killed when very weak. Typical lesions of cholera in both.

TABLE No. 2 — (*Continued*)

Section b. Pork from carcasses that would pass inspection.

Number of experiment	Source of infected material	Quantity fed	Pig number	Date of feeding	Symptoms appeared	Date of death	Remarks
6.....	Flesh and bone from pig No, 106 (Table No. 1).	2 oz.....	11 and 12	Feb. 23	Feb. 28	No. 11 Mar. 4	No. 11 showed typical lesions of cholera. No. 12 developed chronic hog cholera and recovered.
7.....	Flesh and bone from pig No. 107 (Table No. 1).	1½ oz....	13 and 14	Jan. 24	Jan. 29	Jan. 31	Both pigs killed when very weak. Typical cholera lesions in both.
8.....	Flesh and bone from pig No. 108 (Table No. 1).	2 oz.....	15	April 6	April 11	April 14	Pigs killed when very weak. Typical cholera lesions in both.
9.....	Flesh and bone from pig No. 109 (Table No. 1).	1 oz.....	16	Feb. 8	Feb. 13	Feb. 15	Pig killed when very weak. Typical cholera lesions in both.
10.....	Flesh and bone from pig No. 110 (Table No. 1).	2 oz.....	17	April 18	April 22	April 26	Pigs killed when very weak. Typical cholera lesions in both.
11.....	Flesh and bone from pig No. 111 (Table No. 1).	½ oz.....	18	Oct. 27	Nov. 2	Nov. 4	Pig killed when very weak. Typical cholera lesions in both.
12.....	Flesh and bone from pig No. 112 (Table No. 1).	½ oz.....	19	Feb. 1	Feb. 7	Feb. 12	Pig killed when very weak. Typical cholera lesions in both.
13.....	Flesh and bone from pig No. 113 (Table No. 1).	½ oz.....	20	Feb. 1	Feb. 7	Feb. 12	Pig killed when very weak. Typical cholera lesions in both.

Remarks on table No. 2, section a. Experiment No. 1 was conducted in very hot weather. The material fed consisted of rind and subjacent fat. Portions were fed during a period of six days, and, especially in the later feedings, a decidedly rancid odor was present. It is possible that decomposition had something to do with the failure of such large quantities to produce infection. The principal point to be noted is that most of the specimens fed produced hog cholera infection.

Remarks on table No. 2, section b. The experiments recorded in this table were conducted to determine with what regularity fresh specimens from hogs killed while in the early stages of hog cholera, and the carcasses of which would pass inspection, would produce hog cholera when fed to susceptible pigs. Of the eight specimens fed, all produced the disease.

TABLE No. 3

Showing Results of Feeding Refrigerated Pork

a. Pork from carcasses that would not have passed inspection.

Number of experiment	Source of infected material	Quantity fed	Pig number	Date of feeding	Symptoms appeared	Death occurred	Remarks
14.....	Head of femur and flesh. Frozen 20 days.	2 oz.....	21 and 22	Feb. 4	No. 21 Feb. 9	No. 21 Feb. 12 No. 22 Feb. 28	No. 21 killed when very weak. No 22 probably infected from No. 21. Both showed cholera lesions.
15.....	Head of femur of ham frozen 95 days.	2 oz.....	23	April 6	April 11	April 14	Killed when very weak. Typical cholera lesion.
16.....	Head of femur and flesh from ham frozen 62 days. No. 395.	2 oz.....	24	Mar. 25	Mar. 31	April 5	Typical cholera lesions.
17.....	Flesh and bone from virus pig No. 396. Frozen 62 days.	2 oz.....	25	Mar. 25	No symptoms	.....	Pig found April 5 with prolapsed rectum and was killed. Susceptibility not checked.
18.....	Flesh and bone from virus pig No. 397. Frozen 62 days.	2 oz.....	26	Mar. 25	No symptoms	No death	Pig later proved susceptible.
19.....	Flesh and bone from virus pig No. 398. Frozen 62 days.	2 oz.....	27	Mar. 25	April 2	April 5	Lesions of cholera.
20.....	Flesh and bone from virus pig No. 399. Ham frozen 68 days.	2 oz.....	28	Mar. 25	No symptoms	.....	Pig given 3 c. c. of virus, but proved to be immune.
21.....	Flesh and bone from ham No. 423. Chilled 8 days.	½ oz.....	29	May 6	May 11	May 15	No lesions of cholera. Blood proved infectious.
22.....	Flesh and bone from virus pig No. 424. Chilled 8 days.	*	30	May 6	May 12	Did not die	Pig developed symptoms, but recovered. Later proved immune.
23.....	Flesh and bone from virus pig No. 425 Chilled 17 days.	½ oz.....	31	June 11	June 15	June 18	Pig killed when very weak. Lesions of cholera.

24.....	Flesh and bone from virus pig No. 430. Chilled 12 days.	½ oz.....	32	Aug. 3	Aug. 9	Aug. 11	Pig killed when very weak. Typical cholera lesions.
25.....	Flesh and bone from virus pig No. 431. Chilled 12 days.	½ oz.....	33	Aug. 3	Aug. 8	Aug. 11	Pig killed when very weak. Typical lesions of cholera.

b. Pork from carcasses that would have passed inspection.

26.....	Flesh and bone from pig No. 126. (Table 1.) Frozen 58 days.	2 oz.....	34	Mar. 25	Mar. 31	April 7	Lesions of cholera.
27.....	Flesh and bone from pig No. 127. (Table No. 1.) Frozen 58 days.	1 oz.....	35	Mar. 25	April 2	April 7	Lesions of cholera.

*Specimen not weighed. Small button of bone equal in diameter to a nickel, but three times as thick.

Remarks on table No. 3, section a. In this table, the meat referred to as frozen was hung in a rather open garret in an unheated building from the time the animals were killed until samples of their flesh were fed. The weather was such that the hams were frozen most of the time but in some cases there were perhaps a few days during which they thawed to some extent. The meat referred to as chilled was placed in an ordinary refrigerator during the time mentioned.

It is very probable that experiment No. 17 would have proved negative had it been possible to obtain a subsequent check on the susceptibility of the pig fed. Litter mates of this animal were susceptible. Under the circumstances the experiment was classed among those showing undetermined results.

Experiments Nos. 20 and 22 show interesting results. In Experiment No. 20 no visible symptoms appeared and no temperatures were taken. The pig subsequently proved to be immune in spite of the fact that it was a litter mate of seven others all of which were highly susceptible. Thus there is very little doubt that the animal was immunized by the material fed to it. Whether the immunizing effect was due to attenuation of the virus or to the small quantity of virus in the specimen is, of course, unknown. In experiment No. 22 the pig fed showed moderate symptoms but recovered. At one time a temperature of 106° F. was recorded. There is little doubt that it also was immunized in the same manner. Further, it is highly probable that had it been one of a herd of susceptible pigs others would have been infected by associating with it.

In experiment No. 21 the pig fed developed severe symptoms and was killed in order that its blood might be used for virus. A careful autopsy revealed no lesions whatever, so 2 c. c. of its blood were injected into a second pig. This pig developed symp-

toms of hog cholera and showed on autopsy extensive hog cholera lesions so the experiment was classed among those producing positive results. The original pig fed was simply one of those cases, by no means uncommon, in which the disease actually exists but in which its presence cannot be verified by autopsy.

TABLE No. 4

Showing Results of Feeding Cured Pork

Section a. Pork from carcasses that would not have passed inspection.

Number of experiment	Source of infected material	Quantity given	Pigs fed	Date of feeding	Symptoms appeared	Death occurred	Remarks
28.....	Subcutem injections of washings from bone. Ham No. 307.	6 c. c. each	36 and 37	Feb. 28	Mar. 7	No. 36 Mar. 13 No. 37 Mar. 23	Both showed typical cholera lesions.
29.....	Rind from ham No. 307.....	4 oz.....	38	Feb. 28	No symptoms	No death	Pigs later proved susceptible.
30.....	Head of femur and flesh from ham No. 307.	2 oz.....	39a and 39b	Feb. 28	Mar. 4	39a Mar. 6 39b Mar. 10	39a showed lesions resembling cholera. 39b showed lesions of cholera.
31.....	Subcutem injections of bone marrow washings from ham No. 308.	10 c. c....	40	May 18	May 24	May 28	Animal killed when weak. Typical cholera lesions.
32.....	Material from ham No. 308.....	2 oz.....	41	May 18	May 23	May 28	Animal killed when weak. Typical cholera lesions.
33.....	Meat and bone from ham No. 323	2 oz.....	42 and 43	July 15	July 18	No death	Cholera discovered in herd from which pig was taken. Experiment valueless.
34.....	Rind from ham No. 323.....	½ lb.....	44 and 45	July 15	July 20	No death	Cholera of a subacute type discovered in herd from which pig was taken.
35.....	Injection from bone marrow washings. Ham No. 323.	20 c. c....	46 and 47	July 15	.....	.....	No symptoms appeared. Animal later proved immune. Cholera discovered in herd from which pig was taken.
36.....	Injection of bone marrow washings. Ham No. 324b.	10 c. c....	48 and 49	Sept. 30	No symptoms	No deaths	Pigs later proved susceptible.
37.....	Rind from ham No. 324.....	¼ lb.....	50 and 51	Sept 30	No symptoms	No deaths	Pigs later proved susceptible.

38	Injection from bone marrow washings No. 323b	5 c. c.	52 and 53	Oct. 6	No symptoms	No deaths	Pigs later proved susceptible.
39	Material from ham No. 323b	4 oz.	54 and 55	Oct. 6	No symptoms	No deaths	Pigs later proved susceptible.
40	Rind from ham No. 323b	½ lb.	56 and 57	Oct. 6	No symptoms	No deaths	Pigs later proved susceptible.
41	Meat and bone from ham No. 378	2 oz.	58	Feb. 6	Feb. 11	Feb. 15	Pig killed when weak. Typical cholera lesions.
42	Meat and bone from ham No. 378b.	2 oz.	59	Feb. 24	Mar. 1	Mar. 5	Pig killed when very weak. Typical cholera lesions.
43	Meat and bone from ham No. 379	2 oz.	60	Feb. 24	Mar. 2	Mar. 5	Pig killed when very weak. Typical cholera lesions.
44	Meat and bone from ham No. 379b.	2 oz.	61	Feb. 24	Mar. 1	Mar. 5	Pig killed when very weak. Typical cholera lesions.
45	Meat and bone from virus pig No. 413.	½ oz.	62 and 63	July 27	.....	.....	Pigs later proved susceptible.
46	Meat and bone from virus pig No. 414.	½ oz.	64 and 65	June 27	.....	.....	Pigs later proved susceptible.
47	Meat and bone from virus pig No. 415.	½ oz.	66 and 67	June 27	.....	.....	Pigs later proved susceptible.
48	Meat and bone from virus pig No. 440.	1 oz.	68	Oct. 18	.....	.....	Pig later proved susceptible.
49	Meat and bone from virus pig No. 441.	1 oz.	69	Oct. 18	.....	.....	Pig later proved susceptible.
50	Meat and bone from virus pig No. 442.	1 oz.	70	Oct. 18	.....	.....	Pig later proved susceptible.
51	Meat and bone from virus pig No. 515.	1 oz.	71	Mar. 16	Mar. 22	Mar. 28	Typical cholera lesions.

Section b. Pork from carcasses that would have passed inspection.

52	Material from pig No. 152. Table No. 1.	½ oz.	72	Feb. 16	.....	.....	Pig later proved suspectible.
53	Material from pig No. 153. Table No. 1.	½ oz.	73	Feb. 16	Feb. 23	Feb. 28	Pig killed when very weak. Typical cholera lesions.

Remarks on table 4. The cured hams from which the specimens were taken were prepared by a process known as sugar curing. They remained in the brine approximately five weeks, and after being removed were smoked from seven to ten days in green hickory smoke. The brine was prepared according to the following formula:

Common salt	3 pounds
Brown sugar	2 pounds
Saltpetre	2 ounces
Baking soda	½ ounce
Water	4 gallons

Dissolve all the ingredients in the water. Boil slowly for an hour and skim. Allow to cool before using.

This has been selected as a representative formula for sugar curing. There are, of course, many formulæ in use for this purpose, but it is not likely that there is much difference in them as far as their effects on hog cholera virus is concerned. The only substances the use of which the regulations permit in preserving meats are salt, sugar, various vinegars, pure spices, saltpetre and sodium nitrate. Benzoate of soda may also be used, but its presence must be declared on the label, and it cannot in accordance with the pure food law exist in finished food products in excess of 3/10 per cent.

In sugar curing, vinegars are not used and benzoate of soda is used little if at all. Thus the only substances that might be used which do not appear in the above formula are sodium nitrate and pure spices. The former ingredient may be used to some extent in sugar curing processes, and of the spices black pepper is quite frequently used. It is not likely, though, that sodium nitrate exerts more detrimental effects on virus than the corresponding potassium salt, and in the quantities in which they are used in sugar curing it is doubtful if any of the spices operate to kill hog cholera virus.

The outstanding fact brought out in table No. 4 is that the virus of hog cholera in pork is frequently but not always killed during the process of sugar curing. Just what makes the difference between those cases in which it is killed and those in which it is not killed? The three controllable factors involved in the

destruction of viruses by chemicals are the kind of chemical used, its dilution, and the time during which it acts. Can any of these influences be so modified that they will destroy the virus in all cases? This is a question that still remains to be answered.

As circumstances now appear there seem to be no chemicals that could well be substituted for salt and sugar as preservatives. The strength of the brine might be increased, but there is a limit to an increase that would still leave the meat palatable. Increasing the time during which pork is in cure or increasing the time during which it is in the storehouse after being cured may offer possibilities. The fact that the virus was killed in so many of the specimens might seem to indicate that the time limit during which it can survive the sugar curing process was being approached. As a matter of fact, however, there seems to be no definite relation between the time which the hams were in the storeroom and the certainty with which specimens from them would prove infectious. All the hams were in cure approximately six weeks. The time during which different ones were in the storeroom varied from two to eighty-four days. Specimens from the hams representing these two extremes did not prove infectious. On the other hand, specimens from two hams in the storeroom fifty-seven and eighty days, respectively, were found to contain living hog cholera virus. It thus appears that if time is to be employed as a factor in destroying hog cholera virus in sugar cured pork, storeroom cost and interest on money invested must be considerations.

It will be observed that although rind was fed in large quantities in individual cases, no infection was caused by it. It was fed in only three experiments, though, and so few negative results cannot have much significance. In one instance, ham No. 307, feeding the rind did not produce infection, and flesh and bone and also bone marrow washings from the same ham produced hog cholera. In this one instance the virus was evidently killed in the rind when it survived in the deeper parts. Since rind is very likely to find its way into garbage, it is a matter of interest and importance to determine how frequently it carries hog cholera virus, and it is to be regretted that during the time these experiments were in progress scarcity of susceptible pigs prevented deter-

minations of this kind. They are not, though, essential. The real problem is not to determine whether there are parts of a ham that do not contain hog cholera virus; it is rather to determine whether there are parts that do contain it. Bone and bits of clinging flesh are frequently placed in garbage and danger is always present in case they contain virus. It is simply present in a greater degree in case it is found that rind also produces infection.

Besides hams, the parts most frequently sugar cured are shoulders and bacon. There are no good reasons to doubt that shoulders carry hog cholera virus in about the same proportion of cases as hams carry it. It seems quite probable that cured bacon, because of its thinness and because of the relative lack of vascularity of its parts, is less likely to contain virus than are hams and shoulders. This is a point that must be determined with certainty before carcasses showing slight lesions only can be disposed of in the most economical manner.

Viewing the entire situation from the standpoint of biology, a very interesting group of correlated facts is encountered. If the filterable virus were possessed of human intelligence it could scarcely devise a more insidious and ingenious method of self-preservation. It is known to multiply only in the bodies of swine, and conditions favorable for its growth are, therefore, much restricted. Nevertheless, the difficulties met are overcome in a remarkable manner. The virus exists in the blood stream of the animals it infects and is thus distributed to all parts of the body; it cannot at any time be detected with the microscope; it is present in carcasses before gross examinations will detect it; it does not infect human beings, and thus escapes radical measures that would otherwise be taken for its destruction; its presence in herds often drives them to market; it secretes itself in pork where putrefaction, its most deadly natural enemy, is prevented or delayed by curing and low temperatures; then, as a final link in a remarkable chain, the virus, in placing itself where possibilities for its distribution are practically limitless, is at the same time placing itself in material which as a common practice is fed to hogs.

TABLE No. 5

SUMMARY

Kind of pork	Number of experiments conducted	Number positive	Number negative	Number undetermined	Per cent positive	Per cent negative	Remarks
Fresh carcasses that would have been condemned.	5	4	1	.....	80	20	.....
Fresh carcass that would have passed inspection.	8	8	.....	.....	100	.....	.....
Refrigerated carcasses that would have been condemned.	12	8	1	3	88.8	11.2	.....
Refrigerated carcasses that would have passed inspection.	2	2	.....	.....	100	.....	Small number of experiments. Percentage not significant.
Cured carcasses that would have been condemned.	24	9	12	3	43	57	.....
Cured carcasses that would have passed inspection.	2	1	1	.....	50	50	Small number of experimeents. Percentage not significant

In figuring percentage undetermined results are not considered

In general the results shown in table No. 2, section b, should constantly be thought of in connection with those obtained in table No. 3, section a, and table No. 4, section a. The experiments recorded in table No. 2, section b, were conducted to determine whether hog cholera virus in sufficient quantities to infect swine is contained in hams taken from hogs killed while in the early stages of the disease. The experiments recorded in table No. 3, section a, and table No. 4, section a, were conducted to determine the effects of refrigeration and sugar curing on the life of hog cholera virus contained in hams. It seemed desirable in conducting the latter experiments to use hams from pigs known to be infected; otherwise it would not have been known whether negative results were due to absence of virus in the hams before they were treated, or to the fact that the virus was killed during the processes of refrigeration and sugar curing.

The experiments established two important facts: first, hog cholera virus in sufficient quantities to infect swine is quite constantly contained in fresh hams taken from hogs killed before symptoms (other than rise in temperature) appear and before lesions form; second, when specimens were taken from pigs showing lesions, 43 per cent of the cured ones and 88 per cent of the refrigerated ones proved infectious.

Providing all originally contain virus in quantities sufficient to kill, there can, so far as we can see, be no conceivable difference between hams taken from pigs showing lesions and those taken from pigs that do not show lesions, as far as the effects of curing and refrigeration on the virus contained in them is concerned. However, in order to remove doubt concerning this point, experiments were conducted with two cured hams (table No. 4, section b) and two refrigerated hams (table No. 3, section b) taken from pigs showing no symptoms other than elevation of temperature and no lesions. One of the cured specimens and both of the refrigerated ones produced infection. It therefore seems likely that had the hams referred to in table No. 2, section b, been subjected to curing or refrigerating processes, the results would have been similar to those obtained from feeding specimens from virus pigs showing lesions.

When the results of the experiments just described are examined in their relation to practices observed in marketing, slaughtering and inspecting swine there are several phases of the situation that deserve consideration.

Relative to marketing, we are at once brought face to face with the fact that 40 per cent of the pork consumed and 15 per cent of that which is marketed in the country is not inspected. This is killed on farms, by local butchers, and by packing establishments that do not supply an interstate trade. It is a well-known fact that many herds are marketed as soon as hog cholera infection is discovered in them, and in places where there is no inspection, practically all hogs that appear well on foot are killed and sold for food. It is needless to add that large numbers of virus-carrying carcasses must be included among those that reach our markets from these sources. Circumstances thus point to a need for extension of both local and federal inspection.

Turning now to the pork inspected under federal regulations let us examine the regulations themselves with a view to determining how they operate to eliminate from the market carcasses that contain hog cholera virus. First, though, it should be stated that the federal regulations compare favorably with those in force in other countries. The efficiency and thoroughness with which they fulfil their lawful purpose — the protection of human health and human life — is not questioned, but if they do not at the same time operate to protect the swine industry of the country, this fact and the reasons for it should be known, the situation should be looked squarely in the face, and a remedy for it should be sought.

Under existing conditions a consignment of cholera infected hogs reaches market and is first subjected to ante-mortem inspection. With respect to hog cholera, it may contain five classes of hogs: First, dead hogs; these are condemned and tanked. Second, hogs that show undoubted symptoms of cholera; these also are condemned and tanked. Third, hogs that show suspicious symptoms and temperature below 106° F.; these are slaughtered; carcasses that show lesions of hog cholera are condemned or passed for sterilization, according to the extent of the lesions; those that show no lesions are passed for food. Fourth, apparently normal hogs (and those showing suspicious symptoms) that have temperatures above 106°; these are condemned or isolated for further temperature

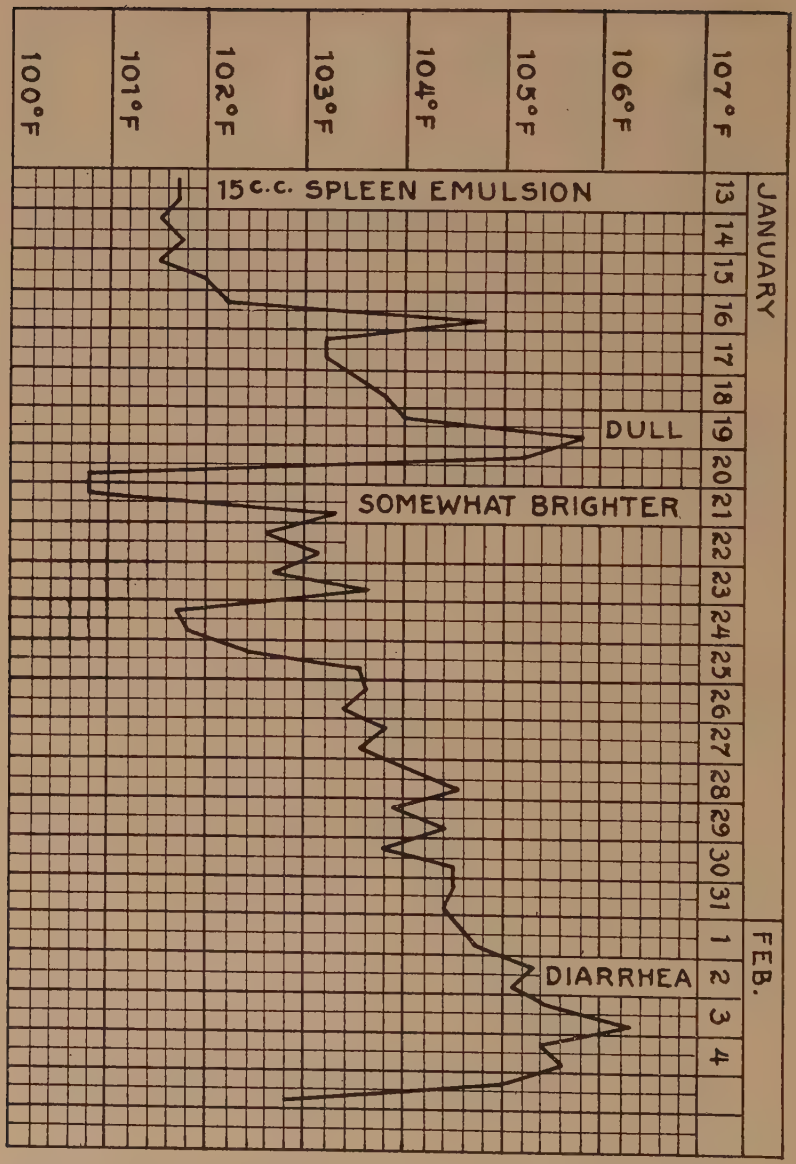
records; in case further temperatures are taken the animals are condemned if their temperatures are still above 106°; otherwise they fall into class three or class five. Fifth, apparently normal hogs that show temperatures below 106° F.; these pass ante-mortem inspection and post-mortem as well if they do not show lesions of hog cholera in organs other than the kidneys or lymph glands.

Briefly stated, the requirements in order that a given hog may pass inspection are that it shall not show undoubted symptoms of hog cholera, it shall not show suspicious symptoms plus any hog cholera lesions, it shall not show a temperature above 106° F., and regardless of ante-mortem findings the carcass shall not, on post-mortem, show hog cholera lesions in organs *other than the kidneys or lymph glands*. What are the chances for virus carrying carcasses to pass inspection? A consideration of symptoms, temperatures, and lesions in their relation to the time at which the flesh becomes infectious, will throw some light on this point.

Relative to symptoms, it need only be stated that a hog will usually show elevation of temperature from one to three days before any marked symptoms of hog cholera appear. The excitement to which hogs are subjected in shipping probably lengthens this time to some extent, because under such circumstances, a slight dullness and sometimes even graver symptoms cannot, even by the closest scrutiny, be detected.

The temperature record, especially when the dividing point is placed as high as 106° F., offers a very uncertain standard upon which to separate infected animals from sound ones but it constitutes a most valuable adjunct to other factors employed for the purpose. In the first place there is a wide variation in the normal temperatures of swine — from 101° F. to 104° F. In the second place weather conditions, excitement due to shipping, and other factors that cannot be controlled alter otherwise normal temperatures very materially. It is very probable that most of these influences when they affect temperatures noticeably, operate to elevate rather than to lower them, and this probably is the reason why the dividing point — 106° F.— has been placed so high. It is certain that some hogs may carry temperatures near 106° as a result of excitement and exertion, and it is equally as certain that many others carry temperatures below 106° when they are suffering with hog cholera.

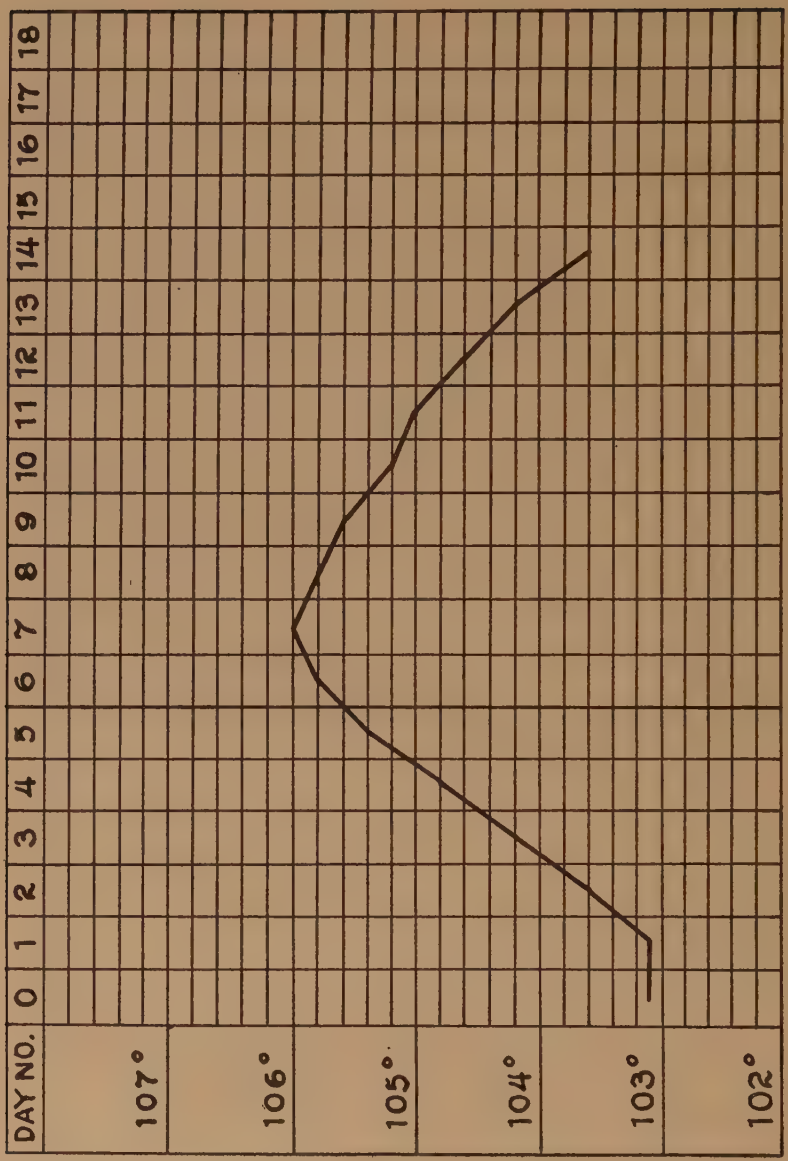
Another important thing to recognize is the *usual* hog cholera curve. It rises quite rapidly, as a rule, remains high for a few days, and then takes a decided drop, which, if death does not ensue, is followed by a second elevation. The following, reproduced from Hutyra and Marek, is intended to show a typical hog cholera curve. It appears originally in the centigrade scale, but it has for the sake of convenience been changed to Fahrenheit.



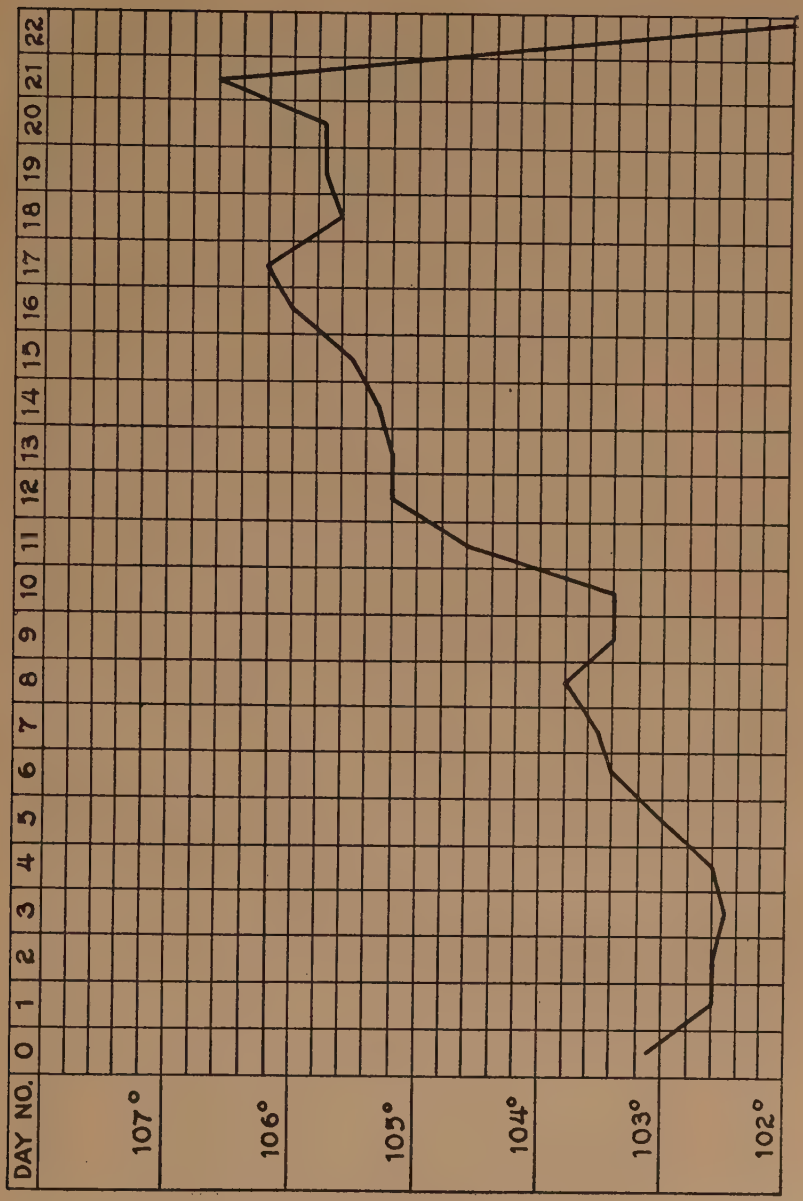
Hog Cholera. Artificial infection with filtered material from a hog affected with cholera. The first rise up to the 4th day of sickness is caused by the primary infection, the second day by the secondary infection. (Hutyra and Marek.)

The temperature curves we record in young pigs usually rise above 106° for a short time, and as a rule they do not fall quite as low between the first and second elevations as did the above curve (January 21). In other ways the curves we record correspond quite closely to the one shown. In this particular case the animal in question would not except during the very last stages of its sickness have been condemned on account of its temperature; symptoms were not recorded until three days after the first decided elevation of temperature occurred; unless the animal was an exception, lesions sufficient to condemn it had not formed during the first day or two on which high temperatures were recorded. Thus there was probably a day or two during which its blood was infectious, when it would have passed inspection.

In order to show more fully something of the number of hogs that will not be rejected on account of high temperatures the following curve, prepared by Craig and Whiting, is reproduced. The animals were infected with intra-muscular injections of small quantities of hog cholera virus. There were 250 of them and the curve shows their average daily temperature during the course of the disease. A second curve, prepared by the same authors, shows the average daily temperature of twenty hogs exposed to cholera by means of natural infection.



Curve showing average daily temperatures of 250 hogs exposed to hog cholera by intramuscular injections of small quantities of virulent hog cholera blood. (Craig and Whiting.)



The above curve shows the average daily temperature of twenty hogs exposed to hog cholera in infected pens. All died of the disease. (Craig and Whiting.)

It will be understood that on any given day many of the temperatures were above and many were below the point indicated. It should also be remembered that there was a period during the time when the curve was ascending when a large number of normal temperatures were averaged with a few that were above normal. In this respect the curves are slightly misleading, but taken as a whole they indicate that during the course of the disease most of the animals showed temperatures below 106° most of the time.

When the lesions are considered as a factor in determining which carcasses shall be condemned it is to be remembered, first of all, that in some cases, even when hogs are allowed to die of cholera, lesions do not form at all. Carcasses representing this class together with those that do not show lesions in organs other than the kidneys and lymph glands are allowed to pass. Hogs do not, as a rule, show marked lesions during the first day or two that elevated temperatures are recorded, and often the time between the first rise in temperature and the time when lesions sufficient to condemn are formed, is of much greater duration. Exemption of the kidneys and lymph glands from consideration unless there are lesions in other organs sufficiently well marked to cause carcasses to be sent to the retaining room, undoubtedly results in passing many virus carrying carcasses.

When table No. 2 is examined in its relation to the symptoms, temperature, and lesions necessary to condemn an animal or carcass for hog cholera, we cannot well escape the conclusion that *there is a time in the life of nearly every hog infected with acute hog cholera when it will pass inspection and when bits of pork from its carcass will prove infectious if fed to other swine.* This time varies from a few hours to several days and is measured, roughly, by the time required for the temperature to rise from normal to 106°, or by the time required for symptoms to develop or extensive lesions to form after the temperature curve starts upward. It is possible that the meat of some hogs is infectious even before the rise in temperature takes place, for it is to be remembered that hog cholera virus *causes* the elevation and it must, therefore, be present *before* the elevation occurs. Whether, or for how long, it is present in quantities sufficient to infect, and before the elevation of temperature occurs, are questions on which we have insufficient data.

Considering again the infected herd as it is unloaded from the car and comparing it with similar herds in the field in which observations have been made and temperatures have been taken, we cannot help knowing that there are often present in such herds considerable numbers of apparently healthy hogs that show high temperatures due to hog cholera. Some of these are weeded out on account of temperatures above 106°, and a few on account of lesions, but many cannot do otherwise than pass. How many, we do not know, but for purposes of comparison it may be stated that during the decade ending in 1911 a yearly average of 18,000 hogs were condemned because of cholera.

Each infected carcass passed possesses almost infinite possibilities in regard to its final distribution. Parts may be worked up into sausage or cooked products, and hams, shoulders and bacon may be cured or shipped in fresh or refrigerated form to supply retail butchers. These facts, coupled with what our experiments have shown relative to the probabilities for the presence of hog cholera virus in market pork, readily lead to the belief that whatever may be the means of spreading hog cholera from herd to herd in different localities, its spread from locality to locality could, if all facts were known, be traced in many cases to shipping and slaughtering hogs in the early stages of cholera and the subsequent sale of pork from the carcasses of these animals.

The results of the experiments described suggest the need of preventive measures for the purpose of diminishing the number of infections due to feeding pork trimmings. These measures naturally fall into three general classes: First, measures to prevent marketing cholera infected hogs; second, measures to turn more carcasses from infected herds into products in which the virus will be killed; third, measures to acquaint swine breeders with the danger involved in feeding garbage containing pork trimmings, and with the ways to avoid this danger.

Preventing the shipment of cholera infected herds should be the first object sought because it attacks the trouble at its source. There will be widespread infection as long as this is a common practice and it will be a common practice as long as it is possible to sell infected hogs for the price that sound ones bring. *Since the discovery of anti-hog cholera serum the breeder has in it an agent which at any given time will usually protect all of his hogs which*

are not, at that time, already dangerous carriers of the hog cholera virus. This statement is based on the facts that pork from hogs killed as soon as an elevation of temperature is recorded proves to be quite generally infectious, and that serum will usually protect hogs treated before an elevation of temperature takes place. Thus it is true that the enactment and enforcement of measures to prevent shipping cholera infected herds need not cause undue hardships in any place where hog cholera serum is available.

The economic difficulties involved in condemning or passing for sterilization infected carcasses which, in reality, are entirely fit for human food, are of a nature which render them very difficult to overcome. The scientific difficulties met in seeking to remove all carcasses that contain virus are no less trying. It has been shown that the carcasses of hogs that show no symptoms other than slight elevation of temperature, and no lesions whatever, may contain hog cholera virus sufficient to infect other hogs. Because the normal temperatures of swine vary so widely no mark can be set that will separate out infected animals with any degree of accuracy. A temperature of 104°, for instance, may be normal or three degrees above normal. There is no method known of detecting all virus carrying carcasses, but, as a general principle, we believe that rigid ante-mortem *herd* inspection, with a more severe interpretation of temperatures and lesions in hogs that are members of infected herds, together with a tagging system rendering it possible to place losses due to condemnation with the man who ships the hogs, are worthy of consideration. Obviously measures of this kind would serve the double purpose of removing more infected carcasses from sale in the form of raw products, and of preventing the shipment of many infected herds that otherwise reach our markets.

Under existing conditions the most promising outlook for dealing with this phase of hog cholera control consists in acquainting swine breeders with the dangers incident to feeding their own kitchen refuse, in case there are trimmings from market pork contained in it. The ordinary farmer has recourse to four very effective methods of protecting his herd from dangers incident to garbage feeding; he may keep pork trimmings out of the garbage, he may discontinue the practice of feeding garbage, he may cook all garbage before it is fed, or he may immunize his hogs. Men who

collect and feed kitchen refuse from cities have recourse only to the two last named methods of protection.

It is sometimes suggested that statutory restrictions should be placed on feeding collected garbage to hogs. The objections to this practice are that it is in a degree repulsive, and that the heavy losses caused by it more than offset the gains it produces. The first objection is well sustained in many individual cases and in others it is not. The French have a saying, "Not what, but how," and this applies well to the point in question. If the material is fed fairly fresh and if the hogs to which it is fed are provided with clean quarters there are no very well sustained objections to the practice, for the material fed is in the last analysis only the refuse from what we ourselves eat. Many thousand hogs are fattened on garbage each year and statutory restrictions placed on the practice as a whole would not, especially since the discovery of anti-hog cholera serum, be justified.

Cooking kitchen refuse to destroy hog cholera virus contained in it is very effective in individual cases, and it possesses the additional advantage of rendering much of the material in it, for instance, potato parings, more palatable and more nutritious. It could not, though, be well enforced as a sanitary measure, it is quite expensive in some localities, and, in order to be effective it requires more time and care than most men will give to it.

Serum-virus immunization seems to be the most logical means of preventing hog cholera in large herds that are fed collected garbage. It is effective, reasonably cheap, and has the decided advantage of protecting from infection by channels other than the one incident to feeding kitchen refuse.

Summary and Conclusions

1. Meat and bone taken from the carcasses of hogs killed before any manifestations of hog cholera other than elevation of temperature take place, and at a time when they will pass inspection, will usually produce hog cholera when fed in small quantities to susceptible pigs.

2. In places where meat inspection is maintained, it is impossible, even with the severest interpretation of temperature, symptoms and lesions now practicable, to remove from market all carcasses of hogs that contain hog cholera virus.

3. We believe a more severe interpretation of temperatures and lesions in hogs known to come from infected herds, will remove many more virus containing carcasses than are now removed, and without resulting in the condemnation of appreciable numbers of carcasses that do not contain virus.

4. The economic difficulties in the way of placing more severe interpretations on temperatures and lesions observed in hogs that are members of infected herds are worthy of study. Whether the number of virus carrying carcasses that pass inspection is large or small, the danger of new infections due to passing them is proportionate to the number passed.

5. In hog cholera infected carcasses that pass inspection:

The virus is not often killed in parts sold as fresh or refrigerated products.

The virus is often killed in hams that are sugar cured. (In our experiments in twelve cases in twenty-one.)

6. Anti-hog cholera serum will, at any given time, usually save all hogs in a herd the carcasses of which will not at that time already prove infectious if small parts are fed to susceptible pigs.

7. Measures to prevent hog cholera infections due to feeding trimmings from market pork should include efforts to prevent marketing infected herds, efforts to prevent the sale of carcasses in products in which the virus is not killed, and efforts to acquaint swine breeders with the danger incident to feeding kitchen refuse.

8. Farmers can avoid the danger mentioned by discontinuing the feeding of kitchen refuse, by placing all pork trimmings elsewhere than in the garbage pail, by thoroughly cooking all garbage before it is fed, or by immunizing their hogs. Men who collect and feed city garbage can avoid the danger by cooking all the material they feed, or by immunizing their hogs.

ACKNOWLEDGMENT.

The writer is deeply indebted to Dr. V. A. Moore whose keen interest in the work has been a constant source of encouragement, and whose advice has been frequently sought and utilized during the four years in which the experiments were in progress.

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HOG CHOLERA AND ITS PREVENTION

R. R. BIRCH

Veterinary Experiment Station

EXPLANATORY NOTE

This article is intended to meet the needs of the practicing veterinarians in New York. In few states, perhaps, owing on the one hand to the large number of New York practitioners, and on the other hand to the relatively small number of hog cholera outbreaks, are conditions more favorable for the handling of this disease by veterinarians only. This, we believe, is a decided advantage to the farmer as well as to the veterinarian. However, the fact that hog cholera serum was discovered so recently as 1908 naturally leads many practitioners to seek information on later developments in serum treatment. A herd of hogs is often worth more than several horses or cattle, and swine breeders more and more are forming the habit of having their animals immunized. It is with the hope of aiding veterinarians to meet this comparatively new demand that this article has been prepared.

The arrangement of material has for two reasons been made with more or less disregard for natural sequence. In the first place it has seemed desirable, so as to avoid any possible confusion, to sharply separate the somewhat complicated laboratory processes from the simpler technique of the field. In the second place the object sought has been to place together in Part II the information most frequently desired by veterinarians. Any practitioner *should* be acquainted with the material in Part I. He *must* from *some source,* if he is to handle hog cholera effectively, be thoroughly familiar with the information contained in Part II.

Finally an effort has been made to bring a new and rapidly developing subject up to date. We know from experience that the recommendations herein contained will, if carefully followed in the field, produce excellent results. We have *tried* to make them the best that can be put forth at present, but to claim that they *are* the best would be folly and egotism.

Special Hog Cholera Number of Cornell Veterinarian. Vol. VI (1916), No. 2.

Part I.

General Remarks

New York is not a great swine producing State, but the hogs within its borders represent a value approximating seven and a half million dollars. The importance of protecting an industry of this size and of providing greater safety for its future development is evident. During the year 1914 the death rate among swine in the United States reached the enormous proportion of 119 per 1,000. During the same year horses showed a death rate of only 20.6; cattle, 19.8; and sheep, 21.7 per 1,000. Ninety per cent of the deaths among swine were due to hog cholera, and must therefore be classed, for the most part, among preventable losses. It is not probable that the disease produces the same death rate here as it does in the Central States, but it is certain that the losses are heavy.

Hog cholera serum protects the swine industry of a state in somewhat the same manner as a fire department guards city property. Three objects are sought in each case: First, to save the immediate property that is in jeopardy; second, to prevent the spread of a plague that may reach uncontrollable proportions; third, to provide safe conditions that invite development. We may profitably carry the analogy a step further and say that the presence of an efficient fire department is no reason why fire-proof buildings should not be constructed, and neither is the existence of a plentiful supply of serum a reason why all possible sanitary measures should not be taken to prevent inter-herd spread of hog cholera. Both safeguards, however, represent great potential value, for each is highly effective as a last resort.

Historical Note

Hog cholera made its first appearance on American soil in the Ohio valley in 1833. Its spread from that time has been quite rapid, aided as it has been in later years by modern rapid transit facilities. The date of its first appearance in New York is not known, but it is certain that the losses caused by it have been greatly underestimated. Very little was *reported* because it was well known among swine raisers that nothing could be done to

check it. Since the discovery of anti-hog cholera serum, however, the number of reports is constantly increasing. This, we believe, is not because of any increase in the prevalence of the disease, but because of a more general knowledge of the fact that serum will prevent it.

Naturally a disease that produces such enormous losses has been the object of extensive researches. The scope of this paper does not permit more than a brief outline of those highly interesting investigations but of the following events each marks an epoch: First, an accurate description of the symptoms and lesions of hog cholera, together with experimental proof of its transmissability, were included in a report of Dr. James Law to the United States Department of Agriculture in 1875; second, Salmon and Smith in 1885 isolated and described an organism (*B. cholerae suis*) which during the eighteen years that followed was quite generally accepted as the cause of hog cholera; third, the discovery by de Schweinitz and Dorset, in 1903, that the disease is really caused by a filterable virus; fourth, the discovery in 1908, by Dorset, Niles, and McBride, of a serum that will protect susceptible swine against hog cholera.

Since 1908 great activity has been manifested in the preparation and use of anti-hog cholera serum. The Bureau of Animal Industry recommended that each state should provide for its manufacture and distribution and thirty states* now own and operate serum laboratories. In addition to these state laboratories many private ones have sprung up, until the money invested in serum laboratories now amounts to more than two million dollars.

Developments in New York. In 1910 the director of the New York State Veterinary College, supported by the opinions of some of the swine breeders of the State, made preparations for the manufacture of serum on a small scale. As hog cholera was not thought to be widely disseminated in the State the plan was to make the project support itself, at least until events justified some conclusion as to the need for more permanent buildings and equipment. The

* Arkansas, Colorado, California, Florida, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maryland, Michigan, Minnesota, Mississippi, Missouri, Nebraska, New York, North Carolina, North Dakota, Ohio, Pennsylvania, Oklahoma, South Carolina, Tennessee, Texas, Virginia, Wisconsin, and South Dakota.

demand for serum increased steadily, the results following its use in the field were, with few exceptions, satisfactory, and in 1913 the legislature appropriated funds for the erection of a serum laboratory and hog house.

In the construction of the laboratory, in its equipment and in its operation, the primary object has been to insure the production of an uncontaminated, potent serum. Sanitary surroundings have been provided for the hogs that must be kept on hand. All the rooms in the laboratory are floored with cement, provided with drains, and the walls are enameled. Steam heat reduces the possibility of dust contamination to a minimum, and a steam sterilizer is installed. A separate room is provided in which the serum hogs are washed and prepared for bleeding. They are then wheeled into a special room and bled, and the blood, immediately after being drawn, is taken to another room to be defibrinated, strained, and bottled. The floors of all the rooms are kept dampened during the time that bleeding is in progress.

Economy of production has been the second consideration. Much modern time-saving apparatus has been provided, but some conveniences which to larger laboratories are necessities, cannot be used to advantage in places where the output of serum is relatively small. Finally, provision has been made for increase in serum production without further expenditures for buildings and equipment. The present output can be multiplied by five with no increase except for operating expenses, and by eight or ten with a small additional appropriation to erect a building for virus pigs.

Serum Preparation

Principle involved. A hog that has recovered from cholera or one that has received active immunization artificially, carries in its blood some unknown substance which protects it from a second attack. This substance does not exist in sufficient concentration to render it possible to use the blood of an immune pig to protect those that are susceptible. The protective strength of the immune's blood must be increased, and this is done by giving it enormous doses of defibrinated hog cholera blood. This process is called hyperimmunization. The defibrinated blood of a hyperimmune

hog, known as anti-hog cholera serum, possesses sufficient potency so that it can be used in convenient doses to immunize other hogs.

Process of serum preparation.[1] Immunize a large hog by giving it 2 c. c. of virus and sufficient serum to protect. After it has recovered from the slight resulting reaction (in ten days or more) it is ready to hyperimmunize at any subsequent time.

Inject a sufficient number of small shoats weighing from forty to one hundred pounds with 2 c. c. each of hog cholera virus. Keep them under observation until they show marked symptoms of hog cholera, and preferably until they show temperatures near or above 106° for two or three days. Kill by bleeding from the carotid, and collect the blood under antiseptic conditions. Reject the blood of any that fail to show marked hog cholera lesions. Defibrinate and cool the blood of those showing good lesions. It is then ready to inject into the large immune animal that is to be hyperimmunized.

Hyperimmunization. Restrain the immune, shave an ear, and disinfect thoroughly. Introduce a hypodermic needle into a prominent ear vein, connect the needle with a pressure bottle containing defibrinated hog cholera blood and allow 5 c. c. of the virus to enter the circulation for each pound the hog weighs. This completes hyperimmunization and the animal is ready to bleed for serum in about ten days.

Bleeding for serum. Restrain the hyperimmune in a portable crate, hose the body well, shave the tail and render it surgically clean. Dampen a piece of gauze in antiseptic solution and cover the body of the animal, allowing its tail to protrude through a small hole in the cloth. Wheel the hog to the bleeding room, remove about one inch of the tail with a sharp chisel, and collect the blood that streams from the wound in a sterile receptacle. (Five c. c. for each pound of body weight is the usual amount drawn.) When the bleeding is finished ligate the tail securely.

Bleeding takes place weekly for three or four weeks after which rehyperimmunization is required. For this a second injection

[1] Four methods may be employed in serum preparation; slow subcutaneous, quick subcutaneous, intraabdominal and intravenous. Only the latter is described as the others are rapidly being abandoned in most laboratories. They require more virus and complications following hyperimmunization are much more frequent.

of one-half the original amount of virus is given. This is followed in about seven days by beginning a second series of three or four weekly bleedings. The final bleeding in which the animal is killed takes place from the carotid.

Handling the blood. Immediately after blood is drawn it is taken to the serum room, defibrinated, and strained through sterile gauze. For each 90 c. c. of blood there is added, as a preservative, 10 c. c. of a 5 per cent solution of phenol. This completes the process of preparation, and the blood, or serum as it is called, is stored in a refrigerator until a test is to be made. Then the combined bleedings from several hogs are mixed in a large container, a sample is drawn for testing, and the remainder is bottled for shipping. The bottles are sealed and returned to the refrigerator until the sample is tested.

Testing the serum. Eight young susceptible pigs, weighing from thirty to sixty pounds each, and preferably from the same litter, are chosen for the test. All are exposed to hog cholera by giving them 2 c. c. each of virus. Two of the pigs receive 10 c. c. each of the serum sample to be tested, two receive 15 c. c. each, and two receive 20 c. c. each of the same sample. Two are given no serum and serve as checks. The pigs are marked for identification, placed together and given like care. Daily temperatures are taken. The requirements of a satisfactory test are that the pigs receiving virus and no serum shall sicken inside of seven days and reach a dying condition inside of fourteen days; and that they shall, on post-mortem, show marked cholera lesions; that the pigs receiving virus and 10 c. c. of serum shall remain well or sicken and recover; that the pigs receiving virus and the larger doses of serum (15 and 20 c. c. each) shall not show any clinical symptoms of hog cholera except a moderate and transient rise in temperature.

A representative test sheet, selected from the book in which the tests are recorded, will be found appended.

PART II

HOG CHOLERA

Cause. Hog cholera is caused by an ultra-microscopic virus which passes through the finest porcelain filters. Its course is sometimes influenced by two organisms, *B. cholerae suis* and *Bact. suisepticum*. Beyond the mere mention of the fact that secondary invasion by the latter organism is usually characterized by pneumonia no further discussion seems necessary. Only on rare occasions does either organism obscure the lesions produced by cholera, and, as a general rule, neither seems to exert a marked influence on the results obtained from serum treatment.

Animals susceptible. Swine of all sizes and ages readily contract hog cholera. Young shoats are most susceptible of all. Sucklings are popularly supposed to be immune but often they are highly susceptible even when only a few days old.

Forms. Three forms of hog cholera are recognized: Peracute, acute, and chronic. The acute form is the one usually encountered. Often an outbreak is ushered in by the peracute form, which as a rule rapidly recedes into acute hog cholera as the disease progresses. Likewise the acute form may gradually assume a chronic nature during the latter part of an outbreak.

Mortality. Acute hog cholera produces a mortality ranging from 80 to 100 per cent. Sometimes in a partially spent outbreak the mortality is somewhat lower and chronic hog cholera develops in some of the animals. Such hogs may live a long while, but as a rule they will neither fatten or reproduce, and are therefore worse than valueless.

Manner of spreading. The filterable hog cholera virus may be carried from place to place in many ways. It is found especially in the urine of infected animals and exceedingly small quantities of it will produce the disease. New purchases, infected streams, animals coming from infected stock yards and cars, and sows taken to infected herds for service all serve to carry hog cholera virus from herd to herd. Horses, cattle, dogs, and other animals, while not themselves susceptible to hog cholera may become the casual carriers of the virus by frequenting or passing through infected

quarters and later visiting yards where sound herds are kept. Sparrows, crows and buzzards also aid in spreading the disease in some localities.

Hogs fed garbage very frequently contract cholera, and experiments conducted at the Veterinary Experiment Station have established the fact that this is due to infected pork trimmings contained in the garbage. Some men, as soon as they discover hog cholera in their herds, at once market all hogs that are apparently well. Some of these animals are really in the first stages of hog cholera, and while their flesh does not harm human beings, it will, when fed uncooked to hogs, produce cholera. Fresh and refrigerated pork and some that is cured will produce infection. Here, then, is a way in which hog cholera virus may easily be carried to remote farms, across the continent, or even beyond the seas.

Period of incubation. Hogs usually exhibit symptoms of hog cholera in seven or eight days from the time they are exposed. Symptoms may though appear in four or five days, and they may in rare cases be delayed as long as twenty days.

Symptoms. The symptoms of peracute hog cholera are obscure. Usually the animal is found dead before it is known to be sick. The acute form usually begins with a rise of temperature, stiffness and soreness. These symptoms are followed, or accompanied, by a disposition to hide in the litter, by chilling, and by a peculiar high-pitched complaining grunt. Conjunctivitis, with resulting sticky discharge from the eyes, is often observed. Constipation usually is present in the early stages and it frequently persists throughout the course of the disease. Often though it gives way to profuse fetid diarrhea. Emaciation as a rule is slight. Refusal of feed, often the first symptom noticed by a layman, is nearly always preceded by some of the above symptoms. Weakness in the hind quarters accompanied by a characteristic weaving gait, convulsions appearing suddenly and lasting a minute or more, and a diffuse purplish discoloration of the skin on the belly, ears and snout, are symptoms that appear late in the course of the disease. Sometimes the skin lesions appear as red or purplish spots and less frequently as ulcers. Cough, thumps and other respiratory and nervous derangements may appear at any time. During the first few days of sickness the temperature is very high, often above

106°. Later a drop from one to three degrees is recorded, and this in turn is followed by an upward trend which may continue until death takes place. Sometimes, though, a second drop takes place and a subnormal temperature is recorded just before death occurs. Rarely does the same animal display all the symptoms described.

Chronic hog cholera is characterized by symptoms growing out of unhealed lesions that occur in the acute form. Thus there is found persistent diarrhea, cough, thumps, deranged appetite and resulting emaciation. The back is arched and the animal appears listless and unthrifty. Frequently there is ulceration and sloughing of the skin.

Lesions. Hog cholera is a septicemia. In the peracute form no lesions are observed. In the acute form the characteristic lesions consist of hemorrhages. The skin may show diffuse hemorrhages on the ears, belly, snout, and at times in the anal and pubic regions. Sometimes small red or purple spots appear, especially on the belly and on the inner surfaces of the legs. Ecchymoses are frequently visible on the heart, especially the right auricle, and the lungs are often sprinkled with petechiæ and ecchymoses. Pneumonia is not infrequent. The red bone marrow is often hemorrhagic. Any or all of the lymph glands may be hemorrhagic, rendering them dark in color, almost black.

Most of the abdominal organs are affected. The surfaces of the kidneys usually show petechiæ, which appear as small, well defined dark red spots, best seen when the capsule is removed. These may also appear in the depths of the organs. In some cases the kidneys appear much darker than normal in color, while in others they are exceedingly pale. The punctiform hemorrhages appear on both the dark and the pale kidneys. The mucosa of the bladder is sometimes dotted with petechiæ. In a typical case of hog cholera the spleen is normal in size or slightly enlarged, and presents on its dorsal surface, usually near the ends or along the margins, black hemorrhagic areas of considerable size. Often, though, it shows no changes except varying degrees of enlargement with corresponding tendency to darker color.

The digestive organs are involved on both the serous and mucous membranes. The lesions on the serosa are by far the most charac-

teristic, and consist of punctiform or larger hemorrhages which may appear on the stomach or any part of the intestines. The ileum and large intestines are most frequently affected. Ecchymoses, less frequently diffuse hemorrhages, and oftentimes erosions are the common changes in the mucosa of the digestive canal. Simple hyperemia is sometimes observed. Hemorrhages are rarely seen in the stomach, are more common in the small intestines, and are most frequently seen in the region of the ileo-cecal valve and the large intestines.

Finally, it is important to remember that many animals die of acute hog cholera without showing any lesions whatever, and negative findings should always be followed by other post-mortems whenever the autopsies are being held for the purpose of making a diagnosis.

In chronic hog cholera the hemorrhages observed in the acute form have usually given way to degenerative, ulcerative or necrotic changes. The intestinal ulcers usually take the form of round, raised, dark colored areas that project sharply from the surfaces of the surrounding mucous membranes. In the older ulcers, rings of dark and lighter color frequently appear on the free surfaces. As a rule the ulcers are situated near the ileo-cecal valve, in the caecum, and in the adjacent colon. The corresponding mesenteric lymph glands are often enlarged, light in color, and indurated. Degeneration of the liver is sometimes observed and broncho-pneumonia is not infrequent.

Diagnosis. An early diagnosis of hog cholera is of the utmost importance if serum treatment is to be used effectively. As there is no quick laboratory method, it is best made by the man in the field. Four things are to be considered: first, history of the case, including evidence of a communicable disease; second, symptoms observed; third, lesions present; fourth, animal inoculation (rarely applicable).

Inquiry should always be made concerning new purchases, the condition of other herds in the vicinity, and concerning other chances for infection. In the immediate herd in question, evidence of the presence of an infectious disease is usually based on the fact that at first only one or two animals become sick, and that some days later other hogs sicken in twos, threes and larger

numbers. In food poisoning, all the animals that are affected usually sicken near the same time, and there is no evidence of the disease spreading from one animal to another. Food poisonings are comparatively rare.

The symptoms are so variable, and so many of them can be produced by conditions other than hog cholera, that, in the beginning of an outbreak, they rarely, in themselves, form the basis of a positive diagnosis. They should always be observed without disturbing the herd, and temperatures of suspicious cases should be taken before the animals become excited. (Chasing a pig to catch it often causes considerable elevation of temperature.) Several exceedingly high temperatures, 106° and over, are, in the presence of other evidence, very suggestive of hog cholera, and may be instrumental in differentiating it from cases of food poisoning. Diffuse purplish discoloration of the skin, when observed in a *live* animal, is in itself almost conclusive evidence of the presence of hog cholera.

The lesions are the most important single factor to be considered, for it is usually possible to base a positive diagnosis on them alone. Like the symptoms, although to a less degree, some of them may be produced by causes other than hog cholera, and a proper interpretation of the post-mortem is highly important. The kidney hemorrhages just described are by far the most characteristic as well as the most constant lesion, and great care must be taken not to overlook them. The capsule should be removed and the surface of the organ carefully examined in a good light. Even as many as three or four distinct punctiform hemorrhages are highly significant, for they may with very little additional evidence furnish ground for a positive diagnosis. The small hemorrhages visible on the various serous membranes, on the lungs, and the somewhat larger hemorrhages on the dorsal surface of the spleen are quite characteristic of hog cholera, but they are seen in a comparatively small per cent of the cases. The same may be said of the petechiæ on the mucosa of the bladder. Causes other than hog cholera will sometimes produce hemorrhagic lymph glands, but these, especially when present in widely separated regions, furnish considerable evidence of the disease. Pneumonia, enlarged spleen with-

out distinct hemorrhagic areas, and the various hemorrhages on the mucosa of the digestive tract, while present in many cases of hog cholera, may often be the results of other causes, so they cannot, either singly or in combination, form the basis of an accurate diagnosis. Purplish discoloration of the skin, so highly significant in live animals, should receive scant consideration in those that are *dead*. Especially in hot weather, a hog dead from any cause whatever may in a few hours present an appearance closely resembling that due to the discolorations caused by hog cholera.

Animal inoculation is rarely of much service on account of the cost involved and the long time required to complete a diagnosis (eight to fourteen days). In case of doubt as to the identity of a disease that continues to spread in spite of measures that usually check hog cholera, and in certain atypical outbreaks that leave doubt concerning the presence of filterable hog cholera virus, inoculation of a susceptible pig with the filtered blood of a sick animal is to be recommended.

Summary. The symptoms most characteristic of acute hog cholera consist of exceedingly high temperatures which are constant and of purplish discoloration of the skin, which is observed much less frequently. Any of the other symptoms described are added evidence. The lesions most characteristic consist of petechiæ on the kidneys, very constant; petechiæ on the mucosa of the bladder, not constant; ecchymoses on the lungs and heart, not constant; petechiæ and ecchymoses on the serous surfaces of the intestines, not constant; black colored, well defined hemorrhages on the dorsal surface of the spleen, not constant; hemorrhagic lymph glands, quite constant but somewhat less characteristic than the other lesions just named.

The diagnosis of chronic hog cholera depends to some extent on the history of the case and the symptoms observed, but it is based more especially on the presence of the familiar "button ulcers" that occur in the intestines.

SERUM TREATMENT

Serum is primarily a preventive of hog cholera, not a cure for it. However, when it is given in increased doses it often saves animals that are in the very early stages of the disease. It is of no benefit to hogs suffering from diseases other than cholera, and for this reason an *accurate diagnosis should always precede its use* in infected herds. There are two ways of immunizing; these are known as the "serum alone" and "simultaneous" methods.

Serum Alone Method. This consists simply of administering the required dose of serum. It produces an immunity lasting a month or more in well herds, and a permanent immunity in hogs exposed to cholera near the time of treatment. It is indicated for the well animals in infected herds, in all cases where a short immunity will meet the requirements, and in all other cases where simultaneous treatment is contra-indicated.

Simultaneous Method. This consists of the administration of a small dose of virulent hog cholera blood in one part of the body, usually the inner region of the ham, and the administration in a different location, usually the inner region of the opposite ham, of sufficient serum to protect against the virus. (See labels for doses.) It produces permanent immunity in all swine except sucklings. Sometimes, though very rarely, an animal will die as a result of simultaneous treatment, and it is probable that others during the process of immunization occasionally excrete hog cholera virus in their urine. Thus the herds in which simultaneous treatment has been administered may at times serve as centers from which infection is spread to other herds. Subsequent litters on the same farm may also be endangered. However, in the immediate herds treated there is no doubt as to the effectiveness of this method. It produces permanent immunity and with little risk.

In general, *the simultaneous method is indicated* in herds where infection is almost sure to occur sooner or later, but where it may be delayed several weeks or months. Such conditions may be present:

First, in sound herds on infected farms. (See discussion of handling an outbreak on the farm.)

Second, where hogs are raised on farms where hog cholera has in the past appeared periodically.

Third, for show hogs under some circumstances. (See discussion of the handling of show hogs.)

Fourth, in very large herds where hogs are constantly being bought and sold.

Fifth, in garbage fed hogs. (See discussion.)

The simultaneous method is contra-indicated:

First, when it cannot be applied by experienced men.

Second, when the entire herd cannot be immunized.

Third, when the herd treated cannot be properly isolated.

Fourth, for suckling pigs.

Fifth, for sows near farrowing time, either before or after.

Sixth, in infected herds. (For exception see discussion of the handling of an outbreak on the farm.)

Seventh, in animals with lowered resistance due to recent shipping, change of food and quarters, weaning, castrating, etc.

Eighth, in all cases where serum alone will be equally efficient.

Confinement of Animals for Serum Treatment. When small shoats are to be treated, an assistant seizes an animal by both hind legs and holds it with its belly toward the operator. In these the site of injection is usually the inner region of the ham, although some prefer to inject in the region behind the elbow. Larger animals, especially pregnant sows, may best be confined by "snouting." A noosed rope, preferably small in size, is placed in the mouth and the noose is drawn tight around the snout. When the rope is fastened to a solid object the hog almost invariably stands quietly and pulls. Animals in the standing position may be injected behind the ear or in the inner region of the hams. Light hogs may be thrown and held on their sides or back to be treated.

Technique of Serum Treatment. Sterilize or disinfect syringe, needles, and a receptacle for serum. Thoroughly cleanse and disinfect the site of injection. (Painting with tincture of iodine is recommended by many.) In injecting in the ham, the needle is

thrust quite deep into the muscle, care being taken to direct the point well back of the femur. In injecting behind the ear care must be taken to insert the needle quite deeply under the loose skin very close to the ear. Injected serum should never raise a well defined welt. The receptacle used to hold the serum should be kept covered except while the syringe is being filled and every other precaution should be taken to prevent dust contamination. In very cold weather it is best to warm the serum near to body temperature, especially when young pigs are to be treated. While serum must always be stored in a cool place it should never be allowed to freeze. An unprotected bottle should never be placed in the sun.

Technique of Simultaneous Treatment. This consists of injecting serum precisely as has been described, and at the same time injecting virus *in another part of the body* with a *separate syringe.* (A tuberculin syringe is convenient for virus as the dose is small — never above 2 c. c.) In small shoats serum is usually injected in one ham and virus in the other. Larger animals may be treated in like manner, or the doses may be administered in the other locations mentioned. Virus is administered in the same manner as serum, but the site of injection should be disinfected both before the needle is inserted and *after it is withdrawn.* All superfluous virus should be destroyed by burning.

Treatment Before and After Immunizing. Preliminary arrangements consist of having the animals placed in dry, clean quarters where they can be caught without chasing them. This should be done the evening before treatment is to be administered. Arrangements on the part of the owner for needed assistants will save time and patience. After treatment has been administered the hogs should be kept in clean quarters, cared for and fed as usual. (Hogs should never be fed so much that they are not hungry for the next feed.) When serum alone is administered in infected herds a second treatment is indicated should new cases appear more than a week after treatment. When simultaneous treatment is administered the appearance in about a week of symptoms suggesting hog cholera should be followed by a second injection of serum alone. If just one or two animals appear dull the second injection should be given to them only. If several are affected the entire

herd may have to be re-treated. This is necessary only in very exceptional cases. As a rule there are no visible after effects when either treatment is administered. A slight and transient elevation of temperature usually occurs in simultaneously treated pigs, but under ordinary circumstances this passes unnoticed.

Reasons for Failure of Serum. First, failure to make an accurate diagnosis.

Second, treating herds too late. Do not expect much in herds where half of the animals are visibly sick. Probably most of the others will show elevations of temperature.

Third, underestimating the weights of hogs. This is a very common error. It is better to treat part of a herd with full doses than to treat an entire herd with part doses. Sometimes a number of pigs left untreated will serve as an object lesson on the potency of the serum.

Fourth, failure to give a second treatment when it is indicated.

Fifth, unknown causes. Sometimes, though very rarely, serum will fail to give protection even when the diagnosis is apparently accurate, and the serum is administered properly. While the causes of these failures are unknown, they must, we are certain, be ascribed to some undetermined peculiarity of the outbreaks in question. The test to which serum is subjected before it leaves the laboratory can leave no doubt as to its potency even when it is used in considerably smaller doses than are recommended in the field. (See description of test in the last paragraph of part 1.)

THE USE OF SERUM IN THE FIELD

General Remarks

In the preceding paragraphs dealing with the indications and contra-indications of serum alone and simultaneous treatment, and in treating the following topics dealing with the use of serum in the field, the writer recommends only such methods as he knows from actual experience will, under existing conditions in New York, produce excellent results. In some sections of the United States, owing to the greater prevalence of hog cholera, to the greater freedom allowed hogs, and to the large number of casual

carriers of the virus (crows, buzzards, etc.) immediate control of the disease has with seeming necessity overshadowed consideration of its ultimate eradication. In these places simultaneous treatment is used almost universally, and it is difficult to see how it can be avoided. Nevertheless, when immediate control can be made to pave the way toward ultimate eradication that is undoubtedly the proper course to pursue. It is with this point in view, and in view of the additional fact that the vast majority of herds in New York never are exposed to hog cholera, that we recommend as a general rule no serum treatment when it can with reasonable safety be avoided, serum alone in infected herds, and a judicious and somewhat restricted use of the simultaneous method in herds seriously threatened. We do this after full consideration of the uniformly good results following the latter method, in the immediate herds treated.

Handling an Outbreak on the Farm. After a positive diagnosis has been made, the attendants of hogs should be cautioned against going to other farms where swine are kept, and against permitting anyone to visit the infected herd. Directions should also be given for the disposal of carcasses. It is needless to disinfect the quarters until all sickness is past.

It is a simple matter to handle, for example, a herd consisting entirely of shoats in which cholera has just appeared, but as every practitioner knows, such easily handled outbreaks are rare. Often there are in other buildings, or pens, swine of all sizes and conditions. In one place there will be a sow and young pigs, in another a number of sows in all stages of pregnancy, and in still another place the herd boar will be found. These animals are neither effectively isolated or positively exposed. How is such a herd to be handled?

Hog cholera is already in the herd of shoats, and in the remaining well ones serum alone will produce a permanent immunity if they are kept together in reasonably close quarters. If they are in a large enclosure it is sometimes best to give all showing a temperature above 104°, and very slight or no physical symptoms, serum alone in double doses, and those showing temperatures below 104° and no symptoms, simultaneous treatment. If a herd is badly infected, simultaneous treatment should not be administered. It is

advisable to take a few temperatures at least even when serum alone is administered. One will often find more infected individuals than the general appearance of the herd indicates, and will thus be more cautious about the prognosis. If new cases of hog cholera continue to appear more than a week after serum has been administered, a second treatment should be given.

The sow and pigs must be handled differently. The sow is often taxed to the utmost to provide milk for her litter and no additional strain should be put on her powers of resistance. The suckling pigs, even if given simultaneous treatment while so young will not be rendered permanently immune. A plan that has given excellent results has been first to give serum alone to both sow and pigs. This will protect them until the effects of weaning are over, at which time all should receive simultaneous treatment. A permanent immunity in sow and pigs will then be the result. Pigs recently castrated and those being weaned may in like manner be protected with serum alone until their physical condition permits simultaneous treatment.

The pregnant sows are a problem in themselves. Under ideal conditions they should be given serum alone to protect any that are exposed, and following this they should be dipped and isolated until after the litters are farrowed and weaned. Then simultaneous treatment may be applied, if it is desired to return them to their original quarters. Unfortunately the practitioner seldom finds ideal conditions and he is therefore constantly obliged to do the "next best thing." When effective isolation is out of the question the sows in early pregnancy should be given simultaneous treatment. The risk due to abortions is slight. Those about to farrow should be given serum alone and they and their pigs should be carried over in the manner already described, until weaning time is past.

The boar is in constant danger of being exposed to cholera, but the immunity produced by serum alone may disappear before infection actually reaches him. Simultaneous treatment is therefore recommended for him and for other hogs on the farm that are not certainly exposed or effectively isolated.

Handling an Epizootic. A widespread outbreak of hog cholera necessitates placing quarantines, attention to shipping regulations,

and various other sanitary measures. With these the State Department of Agriculture is concerned. The part the practitioner plays consists of treating the immediate herds infected, and those that are seriously threatened. He is also frequently asked to advise his clients relative to measures they can take to protect their herds. A client often says to the veterinarian, "Hog cholera is in the vicinity. Should I not have my hogs immunized?" What answer is to be given?

The veterinarian must first determine whether his client's hogs are in much real danger. Have new purchases been made from the infected district? How close is the nearest infected herd? Is it on a stream above the herd in question? Has the client been to see any of the infected herds? These and other questions that circumstances suggest should be asked. The answers will determine the course to be pursued. If the danger appears slight the client should be advised to watch his herd very carefully and report at once any sickness that appears. In the few instances where infection occurs the herd is to be handled in the manner previously described.

Exceptional cases will occur where the danger of infection is imminent, but where some time is likely to elapse before the virus will reach the threatened herd. In these cases simultaneous treatment is indicated. Serum alone would afford protection lasting only about a month, and if infection did not happen to be introduced during that time the herd would again become susceptible.

It is a mistake to apply either serum alone or serum and virus in herds that are not directly threatened. In either case it is a needless expense. When serum alone is applied in a sound herd a lasting immunity is not produced, and often a client will depend on immunity long after it ceases to exist. On the other hand simultaneous treatment, while producing permanent immunity in the animals treated, may be instrumental in infecting otherwise clean quarters, and thus fasten on the swine breeder the necessity of immunizing other animals that births or purchases may add to the herd.

Handling Garbage Fed Hogs. Hogs fed garbage are well known to be in great danger from cholera. In cases where the veterinarian is consulted after the disease has appeared, the handling of the

herd does not differ from that of other herds. Often, though, a man who feeds garbage will recognize his risks and consult a veterinarian before his herd is infected. The chances for infection in such herds are so great that simultaneous treatment of all hogs large enough, and in proper condition, is to be advised. Young pigs and sows not in condition to receive serum and virus should be protected with serum alone until conditions are favorable for simultaneous treatment. All hogs added to the herd should be immunized at once.

Handling Show Hogs. Hogs exhibited at fairs are always in great danger of contracting hog cholera. In addition to the danger due to shipping, the animals must often be quartered with other hogs coming from many localities. These show hogs and the herds they represent are usually of great value, and the advice of the veterinarian concerning precautions to be taken at fair time is often sought. In such cases it should not be forgotten that the *home herd* as well as the *show animals* must be protected. Many a breeder has returned from fairs with his shown hogs apparently well only to have them sicken a short time later and thus become the means of infecting the entire herd. How are the show hogs and the home herd to be protected?

If the show hogs are to be away less than three weeks, serum alone will protect them. On their return they should be dipped and preferably isolated three weeks before they are allowed to mingle with the home herd. If dipping and isolation are impossible the home herd should receive serum treatment *at the time the show animals return.* Hogs that are to be out more than three weeks should either receive a second injection of serum alone at the end of the third week or else they should receive simultaneous treatment *at least a month before they start.* Here again the home herd must be considered. Either the show hogs should be isolated while being given simultaneous treatment and during the four weeks following, or else the *entire herd* should be treated. Even immune hogs may carry home virus on their feet and returning show animals should never be placed with susceptible swine until the former are dipped or the latter are immunized.

Cost of Serum and Virus. Serum is sold at 1¼ cents per c. c., which so far has represented the cost of production. Virus sells

for two cents per c. c. By consulting the labels it will be seen that it costs twenty-five cents to treat a small shoat with serum alone and twenty-seven cents to treat a like animal with serum and virus.

Ordering Serum. Serum is always on hand and is prepared for shipping in three sizes of sealed bottles containing respectively six, twelve, and twenty-four doses of 20 c. c. each. The number and size of the hogs to be treated should first be ascertained. Then the following label should be consulted to determine the number of doses required. Serum is shipped promptly by express unless otherwise requested. Orders received in the morning are shipped in the afternoon. Those received late in the evening are filled the following morning.

N. Y. STATE VETERINARY COLLEGE,
At Cornell University, Ithaca, N. Y.

.....C. C.

ANTI-HOG CHOLERA SERUM

KEEP IN A COOL PLACE

For Intramuscular Injection Only

DOSE:

Suckling pigs	10–15 c. c.
Pigs weighing 50–100 pounds.....	20 c. c.
" " 100–150 pounds.....	25-30 c. c.
" " 150–250 pounds.....	30–40 c. c.

Larger animals in proportion.

Ordering Virus. Virus is perishable and cannot be kept constantly on hand, so it is sometimes, though not often, necessary to wait from one to seven days for it. For this reason it should, whenever possible, be ordered a week or more in advance of the time it is to be used. The name of the veterinarians who will use it must always be given. *Do not use virus without reading carefully the labels on both the virus and serum bottles* that you receive. *Do not give more than 2 c. c. virus to any animal.* The following label should be consulted to determine the amount of virus and serum required:

N. Y. STATE VETERINARY COLLEGE,
At Cornell University, Ithaca, N. Y.

C. C.

ANTI-HOG CHOLERA SERUM

For Simultaneous Use

KEEP IN A COOL PLACE

For Intramuscular Injection Only

DOSE:

Pigs,	40 to 100 lbs.,	20–30 c. c. serum,	1	c. c.	virus.
"	100 to 150 "	30–35 c. c. "	1½	c. c.	"
"	150 to 250 "	35–50 c. c. "	2	c. c.	"
"	250 to 350 "	50–70 c. c. "	2	c. c.	"

Pigs 350 pounds or more, serum in proportion to weight but No increase in virus.

Suckling pigs should receive serum only, 10–15 c. c. This should be followed in 5 or 6 weeks with the simultaneous treatment.

Serum and virus must both be stored in a dark, cool place. A refrigerator is the most desirable place to store them but the use of either a cellar or a basement is permissible.

References. For other publications covering about the same ground as is covered in this paper, and describing various practices observed in using serum in the field, the readers of the CORNELL VETERINARIAN are referred to many excellent bulletins published by the various states that prepare and distribute serum. Among the states that produce large quantities of serum may be mentioned Iowa (Iowa State College, Ames); Ohio (State Serum Institute, Columbus); Kansas (Kansas State Agricultural College, Manhattan); Missouri (Veterinary Department, Missouri University, Columbia); Indiana (Agricultural Experiment Station, Purdue University, Lafayette); and Tennessee (Cholera Serum Department, 1502 Clinton street, Nashville).

For various publications relative to the development of hog cholera serum and for important data dealing with the economic significance and control of hog cholera, reference is made to the recorded work of the U. S. Bureau of Animal Industry.

For a more inclusive and detailed discussion of hog cholera and hog cholera serum, reference is made to the last report of the A. V. M. A. committee on diseases. (Moore, Dimock, Haring, Gilliland, Kinsley.) This appears in the Journal of the American Veterinary Medical Association, November, 1915.

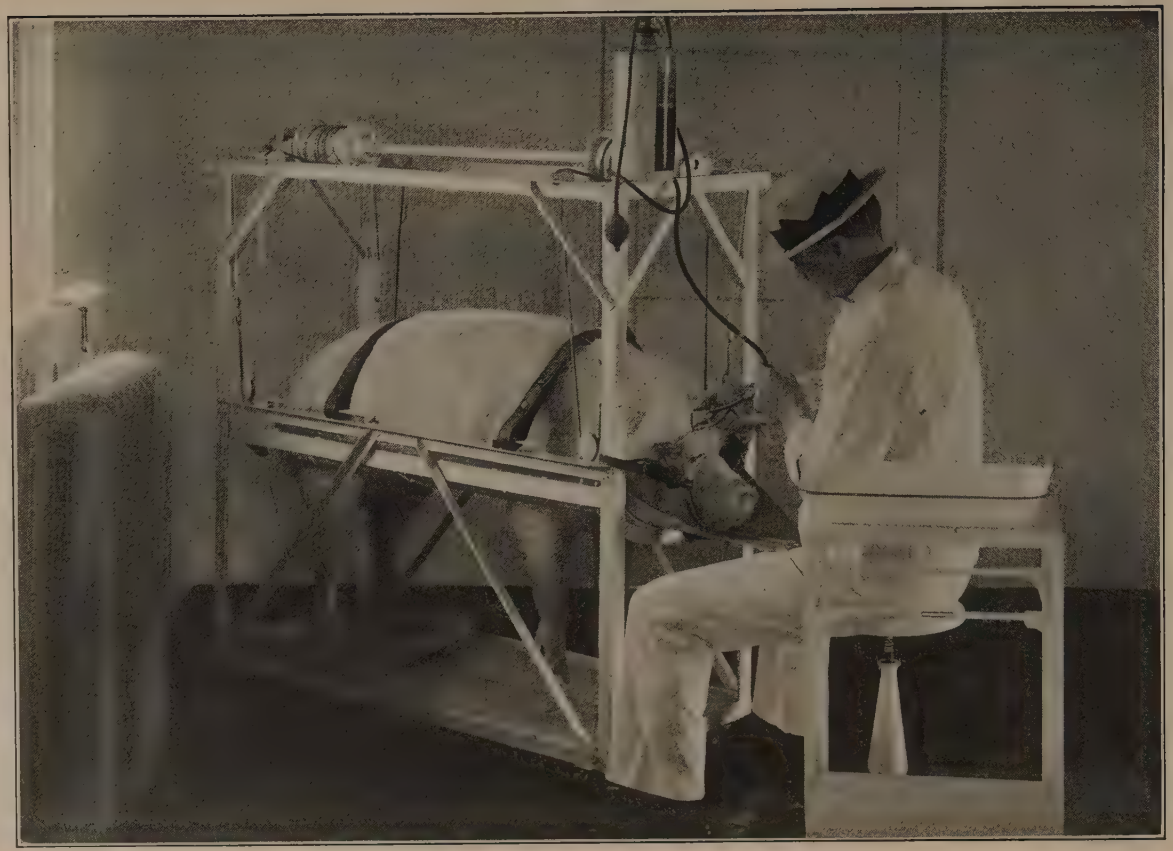
Serum laboratory and hog house.

Interior view of hog house.

PLATE III

Pigs affected with acute hog cholera.

PLATE V



Hyperimmunizing. Showing crate for holding pig and method of injecting the virus.

Tail bleeding for serum.

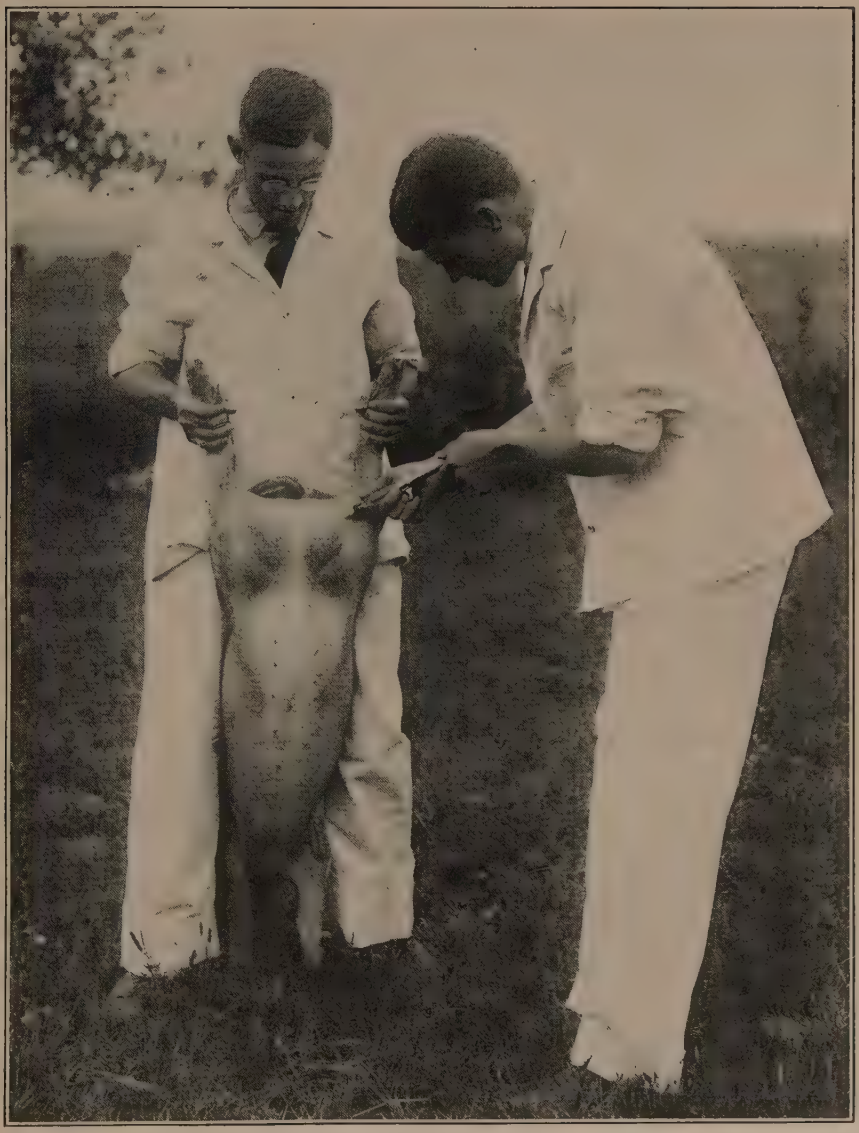
Serum laboratory. (Straining serum.)

Injecting serum behind the elbow.

PLATE IX

Injecting serum behind the ear.

PLATE X



Injecting serum in inner region of thigh.

RESEARCHES UPON ABORTION OF CATTLE*

W. L. Williams

Department of Obstetrics and of Research upon the Diseases of Breeding Cattle

Our researches during the past year may be outlined as follows:

(1) A continuation of studies of the genital organs of cattle upon the killing floors of abattoirs.

(2) A continuation of our studies upon the small experimental herd belonging to this department.

(3) A continuation of our studies upon the large dairy herd designated as Herd B.

(4) A study of the pathology and handling of calf scours and pneumonia.

The work of this department, as well as that of the Department of Surgery, was handicapped during the academic year of 1914–1915 owing to the two departments having but one assistant. The incumbent, Dr. J. F. Shigley, extended every possible aid to this department, as well as to the Department of Surgery, and rendered much very valuable service.

Assistant Professor C. P. Fitch has rendered highly valuable services in pathology, bacteriology and serologic tests.

Owners and superintendents of large and highly valuable herds of dairy and breeding cattle have co-operated in a very generous manner in the furnishing of valuable data, and also, when animals have been found incurably sterile, have made it possible for me to procure the diseased genital organs after the slaughter of the animals, thus adding to our already highly valuable pathologic collection.

The trustees of Cornell University asked the Legislature for a special appropriation of fifteen thousand dollars to be devoted to researches upon cattle abortion. The breeders of pedigreed cattle, having learned that the appropriation had been asked, gave the bill

* The Bureau of Animal Industry, U. S. Department of Agriculture, co-operating.

their vigorous support with such earnestness that it was granted virtually, if not wholly, without dissent. Their intelligent appreciation of the magnitude of the problem and its economic and sanitary importance to the people of the State enabled them to advise their representatives in a convincing manner of the value of the legislation. It is to their intelligent and vigorous support of the measure that we are indebted for its passage. Unavoidable delay in connection with the building of suitable stables has occurred, but the plans are matured and it is fully expected that suitable quarters for the researches upon a larger scale will soon be available. Meanwhile the experiment herd already on hand is being used to the utmost and has increased materially in size.

With the new department definitely established, an assistant, with the grade of instructor, has been furnished, and Dr. W. A. Hagan, a young man of excellent preparation and with valuable experience, is now devoting his entire time to this work. Assistant Professor Fitch, of the Department of Bacteriology, is also co-operating in the work, conducting and supervising in a general way the bacteriologic and histologic side of the problem, and is entering with enthusiasm upon some extended and special studies of the ovaries of cows.

We are taking up some interesting serum experiments upon heifers in first pregnancy. Our researches have been varied. Some parts of the work have not reached sufficient maturity to warrant recording.

The diagnosis of contagious abortion has not yet been placed upon a secure basis. Some cases are so evident clinically as to leave little doubt in the minds of most persons, but the clinical diagnosis is based largely upon the ratio of abortion, evidently an insecure standard. If, in a herd of one hundred pregnant cows, one aborts, the evidence of contagion is just as good as would be the evidence of each one of fifty cows, should that many abort, but the diagnosis is usually based upon the collective evidence of the entire fifty cases. If an animal is destroyed immediately after aborting as a result of contagion, there is found in the utero-chorionic space the "exudate of contagious abortion," a thoroughly characteristic substance, yellowish or greenish brown, of a soft, putty-like consistence, very adhesive, and containing great numbers of abortion bacilli. Ordinarily, substances do not adhere

readily to the uterine mucosa or to the exterior of the chorion, but this exudate adheres very firmly to each of these tissues. This adhesiveness detains the exudate, and in it the bacteria, so that it extends by growth rather than by moving in obedience to gravity or through transfer by other agencies.

Sometimes this exudate may be recognized clinically prior to, during, or after aborting or calving. The exudate, usually mingled with blood, is expelled from the vagina in varying amounts, usually mixed with fluids which tend to break it up into a flaky mass. After parturition or abortion, the presence in the uterus of the infection of abortion is often revealed by a scarlet discharge, usually of a creamy consistence, the scarlet being mixed with yellow flakes, giving to the mass a dirty appearance. As the mass exudes, it collects on the tail and buttocks, where it quickly blackens as crusts of blood. Some writers regard this as "normal," in the sense that it is common, but it must be recognized, in the light of recent findings, as the specific discharge of contagious abortion.

The retention of the fetal membranes is rapidly becoming accepted as contagious abortion. Technically, any infection which may gain entrance to the pregnant uterine cavity may cause retained fetal envelopes; practically the only known organism capable of such primary invasion in the cow is the abortion bacillus. After the abortion bacillus has invaded the uterine cavity and the uterine seal has been prevented or broken down, other organisms (colon, streptococci) may and do invade the cavity, to complicate and intensify the disease. Clinically, as well as technically, so far as researches have gone, from a biologic standpoint, the presence of retained fetal membranes justifies abundantly the diagnosis of contagious abortion.

Premature birth, so far as has been investigated, is always the consequence of contagious abortion, and the infection is approximately equal in the dam and her progeny.

Sterility is due chiefly to contagious abortion, but a given case of sterility can not readily be so proven. The diagnosis is made largely upon general clinical observations and deductions from clinical facts. In the sterility of contagious abortion there are apparently two types — primary, due to the direct action of the abortion bacillus; and — far more numerous — secondary, as the

result of the secondary invasion of the genital system by other organisms.

The scours and pneumonia of calves are due at times primarily to the abortion bacillus, but are almost always quickly complicated by the invasion of colon and other organisms. It appears probable that many cases are due primarily to the colon or other organisms without the presence of the abortion bacillus, but this is difficult of verification at present. The growth of the abortion bacillus is very slow and weak. It is difficult of identification in the presence of more luxuriantly growing bacteria.

The serologic tests (agglutination and complement-fixation) are inaccurate tests of individuals, and are not yet upon a secure foundation. The basic defect in the entire problem of diagnosis is the absence of a proven lesion of universal or even general occurrence. Abortion itself can scarcely be recognized as a lesion and only with reservation as a symptom. Abortion can be caused by douching the pregnant uterus and in other ways. The phenomenon of abortion is not the lesion, however, but the culmination of a series of lesions. The same holds true of the other means for diagnosis: each has its limitations. In other chronic diseases, there are recognized definite and pathognomic primary lesions. In tuberculosis the specific lesion, tubercle, has played the title role and was long the basic diagnostic factor. In glanders, also, there is a specific nodule which serves to identify the disease. Even in contagious abortion of cattle, we recognize a specific lesion in the guinea pig, the nodes of contagious abortion in the liver and spleen, but in cattle no such lesions are yet recognized. Accordingly, when the serologic tests are attempted, there is no available check which makes a secure foundation. The blood of a cow may agglutinate high, but no lesions are recognizable, and perhaps the bacillus can not be found. The diagnostician must resort to deductions based upon insecure foundations. It is known, for example, that the blood of a majority, but not nearly all aborters, will agglutinate at 1–100 and many of them at 1–1000 or upwards. So it is arbitrarily decided by many that if the blood of a cow agglutinates at 1–100 (some say 1–50) she is or has recently been infected with contagious abortion. As a rule, if the blood of a cow agglutinates at a lower rate than 1–50, say at 1–25 or 1–10, she is not very

likely to abort, have retained afterbirth, or show other marked disease. Many class agglutinations at 1–25 or 1–10 as negative, but there is no definite evidence that such animals are free from the infection. Indeed, there is much evidence that they are not free. On the other hand, the blood of cows which abort, calve prematurely, or have retained afterbirth due to contagious abortion may at the time of the disaster be negative at 1–25 or less. Hence, the serologic tests, highly interesting and valuable in many respects in research work, are of scant practical value in determining the status of an individual in connection with the control of the disease.

The abortion bacillus rarely grows in pure culture in the bodies of cattle, so far as yet determined. The chief, if not the sole location yet determined is the utero-chorionic cavity in the pregnant uterus. There, when the exudate of contagious abortion is present, the bacillus is ordinarily recognizable in smears and by artificial cultures. In the udder, where the bacillus is common, it is associated with a variable number of other bacteria, largely more profuse growers. When cultures are attempted, the bacteria, which grow more rapidly, tend to veil the abortion bacillus and interfere with its recognition, thus lowering the diagnostic accuracy of such search. Elsewhere in the bodies of cattle, except occasionally in lymph glands centripetal to the udder (Schroeder), identification of the abortion bacilli in adult cattle is extremely rare. Whether it is absent or whether not recognized because of imperfect technic, is yet to be determined. Experimental inoculations of guinea pigs with suspected milk or other tissues or fluids, while of experimental interest and value, are subject to serious limitations as a diagnostic process.

The present methods of diagnosis are accordingly each subject to important limitations. When contagious abortion is severe, whether in the aspect of abortion, sterility, retained afterbirth, or calf scours, it is readily diagnosed, but as the severity wanes and we approach the border line of health, the traces become indistinct and confusing and the actual line of demarcation between infection and non-infection is not recognizable by any test now known. That line will probably not be made clear until a fundamental lesion, uniformly following infection, is recognized.

THE AVENUE AND DATE OF INTRA-UTERINE INFECTION

Uterine sterility, abortion, premature birth and retained fetal membranes can be caused only by intra-uterine infection. It was assumed originally, quite naturally, that such invasion takes place exclusively through the vulva and vagina. Later evidence was submitted tending to show that the infection may invade the bodies of adults through the digestive tract, in the form of contaminated herbage or grain, but the evidence failed of completion, leaving the question somewhat open. Later the recognition of the abortion bacillus in the milk by Schroeder and Cotton, Evans and others showed an essentially obligatory infection of the new-born calf through the alimentary tract due to contaminated milk. The presence of the infection in young calves has been recognized microscopically, culturally, and serologically. By the serologic tests, the infection has been followed up to first pregnancy and abortion. The transfer of the infection from the digestive tract to the uterus has not been made wholly clear. Two possibilities arise. The infection may escape per anum to re-enter the vulva almost immediately, but there is no evidence that this occurs, and recent developments throw great doubt upon any such probability. The second, and now apparently certain course, is that the bacillus enters the blood stream from the intestine and is carried thence to the genital tract.

The evidence thus far submitted of the power of the bacillus to penetrate the healthy intestinal mucosa of adults and gain the blood stream is very slight, but in new-born calves the evidence is strong and convincing. All recorded observations of the invasion of the fetus show, where any indications occur, that the fetal invasion takes place through the alimentary tract. Frequently the bacillus is found in the stomach and intestines without being recognized in the fetal blood or glands, but never in the fetal blood except it is present in the alimentary tract. This indicates that the primary invasion was through the chorion into the amniotic fluid. The fetus swallows its amniotic fluid constantly. As the fluid is contaminated, alimentary invasion is inevitable. After the calf is born, alimentary invasion becomes inevitable when the milk it is fed is contaminated. When the exterior of the udder and teats of the cow is contaminated by discharges from the geni-

tal tract, the calf gets the infection when sucking the contaminated teat. The genital discharges contain colon and other dangerous organisms associated with contagious abortion in the uterus and with scours in the new-born calf. The scours causes lesions of the intestinal epithelium, facilitating greatly the penetration of the mucosa by the mixed infection. In this manner the infection reaches the blood stream. Thus the calf is exposed through the ingestion of milk highly infected within the udder and yet more highly infected from the exterior of the udder due to soiling by the intensely virulent emanations from the genital tract. The exposure of the young calf is not for one meal or one day, but is continuous so long as it is allowed to suck or is fed upon raw milk, while if the enteritis of scours is present there is increased opportunity for the infection to reach the blood stream. Thus the calf, more susceptible to infection than the adult, is continuously exposed to large volumes of infection of a highly virulent type, while the far less susceptible adult is hypothetically exposed to possibly conaminated food at unknown intervals. Once in the blood stream, the infection is carried to and apparently into the cavity of the genital canal, to cause disaster.

Schroeder contends and presents evidence to indicate that the infection may also enter through the udder. He emphasizes as the general danger at this portal the transfer of the bacillus by the hands of the milker from the infected udder of one cow to the non-infected udder of another cow. This danger can not be operative, or at least only technically so, in heifers pregnant for the first time, where contagious abortion exacts its heaviest toll. Neither is this avenue an important peril to cows whose udders are already infected — and it is at present quite unknown how many or how few udders of milk cows are free. There is no great reason for assigning a preponderant role to the transmission of the infection from udder to udder by the milker. If the milk is infected, the calf must become infected. The contention of Schroeder that the udder is the most constant and perhaps most permanent reservoir of the abortion infection may be freely granted. It does not follow, however, that the udder gives off (in the milk) the largest volume of infection, or the most virulent infection. In the latter respects, whether viewed from the stand-

point of clinical observations or post-mortem findings, the diseased uterus excels. We have observed as much as three or four gallons of the exudate of contagious abortion, teeming with abortion bacilli, in one uterus. It is scarcely conceivable that one udder or many udders could contain an equal volume of infection. Clinical observations indicate, as is shown by the researches in Herd B, that the infection from the exterior of the udder, which has flowed down the tail and buttocks, is by far the most important source of infection.

THE IMMUNITY OF CONTAGIOUS ABORTION

It was stated in 1912 that there is no natural immunity acquired in contagious abortion such as is observed in most acute contagious diseases, like rinderpest and foot and mouth disease. Abortion is a chronic disease. The outstanding difference between an acute and a chronic infection is in their power to produce immunity. In acute infectious diseases there is an immunity against invasion; in chronic infections there is an immunity against the disease-producing power of a persisting infection. Perhaps that thought can be well illustrated by the use of the chart of eighteen heifers recorded in 1912, under experiment with abortion bacterins, bringing the history of the group down to date. As recorded in 1912, each of the 18 heifers received four doses of the then very popular abortion bacterins, in order to test their power to prevent abortion.

CHART I

Vital Statistics of 18 Heifers in Herd A, given Abortion Bacterins in First Pregnancy, with Female Progeny.

Number	Born	1912	1913	1914	1915	1916	Remarks
1	12.6.09	A					Destroyed 10.9.12. Gangrene of the Uterus. Decomposition of Foetus
2	10.6.09	A					Died of Metritis
3	2.24.11	S or A	UNSEEN				Slaughtered on account of Sterility
4	10.30.10	S or A	UNSEEN	A			Slaughtered on account of Sterility
5	9.6.10	A	A	S			Slaughtered on account of Sterility
6	9.29.10	X	B	(B H (*6A))		B	Slaughtered on account of Sterility
6A	11.8.11			*		B	
7	11.6.10	A	(Twins)	D			Died of Metritis
8	4.26.11	H (*8A)					Slaughtered on account of Sterility
8A	12.8.12	*		A			Slaughtered on account of Sterility
9	7.20.10	B					Retained afterbirth. Sold on account of inefficiency
10	1.1.11	B	B				Sold account of Bad Udder - Gangrene - Half Amputated
11	2.5.11	S or A	B	B	B		Sold account of Low Dairy efficiency
12	9.22.10	A	H (*12A)	B			Died of Indigestion - 1915
12A	11.26.13				H	H	
13	10.5.10	H (*13A)	B	H	B		Sold – Efficient
13A	11.27.12	*		A	H	B	
14	10.20.10	H (*14A)		B	B	B	
14A	12.9.12	*			H		Sold - Efficient
15	10.18.10	B	H		B B		1913 Heifers died 2.10.14
16	10.5.10	A	B	B	B	H	
17	10.5.10	A	H (*17A)	B	H (*17B)	H	
17A	9.10.13			*	B	S	
17B	9.8.15				*		
18	12.9.10	A	H (*18A)	H	H	B	1914 Heifer died from Indigestion
18A	12.31.13		*			B P	

Died or Killed	10
Sold in Breeding Condition	5
Remaining in Herd of Dairying Age	11

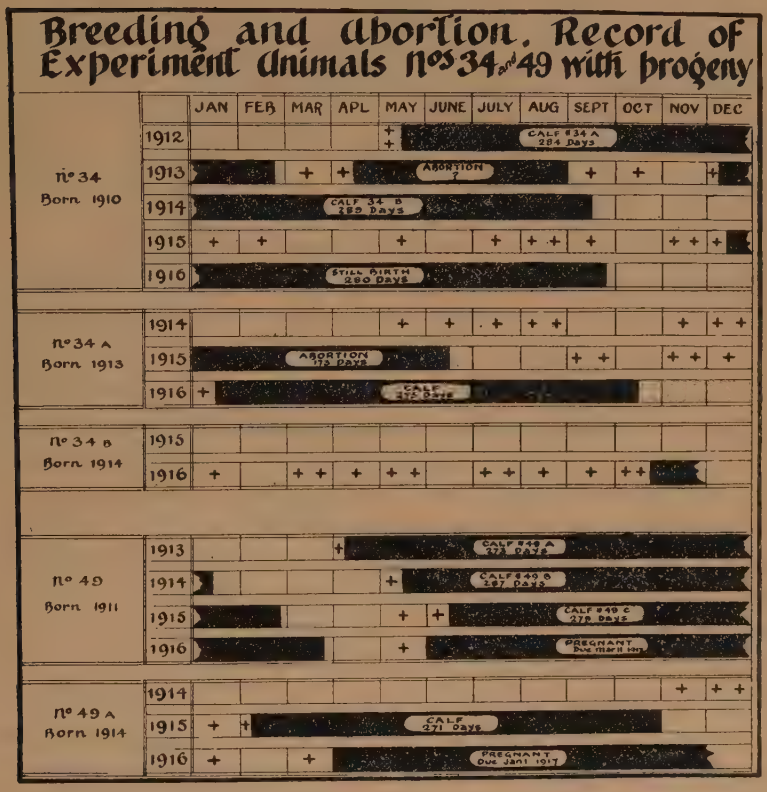
A, abortion
X, premature birth
S, sterility
B, bull calf

H, heifer calf
P, pregnant
D, new-born calves died

Assuming that each of the 18 heifers should have calved once a year and that one-half the calves would be heifers, there should now be in milk, counting the 18 heifers, their daughters, and 4 granddaughters, a total of 49 females. Instead, there exist in the herd 11 females of dairying age. Five cows, presumably capable of breeding, have been sold, and 10 have died or been killed. In short, the size of the original group has been diminished by 5 animals, or 28 per cent, counting in the group all the female progeny of dairy age. This certainly indicates that no valuable immunity is caused by severe infection. Incidentally also the chart tends to negate the contention of those now claiming to prevent abortion by hyperinfection prior to conception. These were certainly hyperinfected when two years old, and have been liberally infected ever since. If that would prevent disaster in later years, this group should have become highly valuable.

The principle which I wish to bring out may be further illustrated by Numbers 34 and 49 of our research herd. Each was purchased at birth and has been under constant observation up to the present time.

CHART II



Copulation with the bull is indicated by +, and pregnancy, when verified by rectal and vaginal palpation, is indicated by filling in the spaces between ruled lines with black. Blank spaces between + marks indicate that for some undetermined reason (embryonic abortion, retained *corpus luteum*, etc.) the animal failed to come in heat.

Number 34, a strong heifer, apparently well, was inoculated in the jugular vein during her first pregnancy with a large volume of abortion cultures. She gave birth at full term to an apparently healthy calf, our Number 101. She then conceived with difficulty, aborted two or three times unseen, and finally gave birth to a healthy calf, our Number 3. After much difficulty, she conceived again, to abort on September 20, 1916, at the 280th day of pregnancy. Following this she almost died from metritis. Her afterbirth was retained for eight days and when it came away all the cotyledons came with it. Her first calf, Number 101, in utero

when 34 was inoculated, born 1912, was caused to conceive only with very great difficulty. Apparently she aborted once or twice unseen. Then she conceived, to abort at 173 days. She finally conceived again, to expel prematurely, upon the 276th day of pregnancy, a heifer calf weighing 65½ pounds. Her placenta was retained, and, as in her mother a few weeks before, the cotyledons became necrotic and sloughed away. It required the best attention we could give them to preserve their lives. Whether they will ever breed again, is a wholly different question.

The heifer calf of Number 101 was evidently ill at the time of birth. She was dull and appeared in a stupor; she lay down most of the time and refused to eat; her meconium, or fetal feces, was swarming with bacteria; her large intestine was one great cesspool of infection. After vigorous handling with calf scours serum, she rallied and began to grow. She had the infection abundantly when she was born.

The second heifer of Number 34, our Number 3, born in September, 1914, has given great trouble in breeding. She has apparently conceived twice and aborted unseen. Her genital organs are normal, as far as physical examination reveals, but she simply refuses to become pregnant.

Number 49 has been served five times, has produced three healthy calves, and is due to calve in March, 1917. During her first pregnancy, she was fed a liberal amount of pure cultures of abortion bacilli. Since 1913 she has been the constant companion of Number 34. She has also been in the same stable — and in an adjoining stall with an open partition — with the aborter Number 101. She has run in the paddock with her. One of her mates of the same age aborted in the pasture where she was grazing during her first pregnancy. She has been exposed and re-exposed, naturally and experimentally.

The first heifer of Number 49, our Number 1, bred promptly and calved perfectly before she was two years old. The calf, Number 11, is strong, well developed, and healthy. Number 1 conceived again very promptly and is due to calve on January 1. The blood of Number 34 has always reacted high; the blood of Number 49 has always reacted low. The blood tests of the calves of 34 have averaged very high; the blood tests of the calves, even

including the second generation, of 49, have always been low. Number 34 and her daughters have always given great trouble in breeding; Number 49 and her heifer have bred promptly.

Number 49 has withstood all the exposure of Number 34, except that her experimental infection was given per mouth instead of in the jugular vein. She has suffered from no serious disaster and has to-day a higher power of resistance than 34. In other words, the exalted power of resistance — call it immunity or what you will — has been acquired, not through disaster, not through abortion, sterility or retained afterbirth, but through the avoidance of these.

Researches in Herd B

Herd B is a large dairy herd used for the production of certified milk. It had already been operated several years when it was removed to the present location, upon an estate of several hundred acres, where it is aimed to produce sufficient ensilage and hay for the herd and part of the bedding and grain.

Prior to moving into the present quarters, the herd had become badly contaminated by tuberculosis, and under semi-annual tests, many had been destroyed. On March 3, 1909, when the herd was moved to its present location, it consisted of 876 animals of which 504 were in the dairy, 86 were pregnant heifers, and 286 were heifer calves and unbred heifers.

It has been the plan to milk 500 cows. Originally the herd consisted of registered Jerseys, but subsequent to 1909 there have been added at various times grade Jerseys and Ayrshires. Throughout the history of the herd, except for a brief interval from February 5 to September 1, 1912, it has been aimed to grow all heifer calves born in the herd. In the interval named, 118 heifers were sold for veal. Except for these, no heifers have been discarded, except because of death or disease.

The herd has always been free from acute infectious diseases. There have been no losses, or at least no material losses, from anthrax, blackleg, hemorrhagic septicaemia, etc. No efficient dairy cows have been sold. Instead, there have been bought at intervals 627 cows in an effort to keep the milking herd up to the desired 500. Twenty-four per cent more cows of dairying age were added by purchase from May, 1909, to January, 1915, than

were in the herd when brought to the present farms. Ideally each female of dairying age should give birth to one calf each year, and the sexes should be approximately equal in numbers. Starting with the original 504 dairy cows, there should have been each year 252 heifers per annum or 21 per month. Estimating the dairying life of a cow at six years, that is, an age of eight years, there would be an average annual loss by death and discard for inefficiency of 16 2/3 per cent, or in a herd of 504, 83 cows, which should leave a surplus of 169 cows instead of a deficit of 110 cows per annum, as has occurred, a difference of 279 cows.

As in any herd, there were occasional losses from accident and miscellaneous diseases and considerable losses because of diseased udders. There were many cows of low efficiency held at a loss in an endeavor to keep up the total milk yield. The deaths and discards were so serious that it was only with extreme difficulty that the milk yield of the herd was maintained at something near the minimum requirements. There was accordingly no opportunity to cull with a free hand.

The notable losses in the herd belonged to the two great groups of tuberculosis and the abortion-sterility-calf scours group.

Although Herd B was seriously tuberculous and the losses therefrom enormous, they have not equalled those from the abortion-sterility-calf scours group. The ravages from tuberculosis were, however, more conspicuous. The losses from the abortion-sterility-scours group were individual; a cow or a calf disappeared and her place was taken by another. The tuberculous animals were destroyed in large semi-annual groups, which was very impressive. It is far more spectacular to see 100 animals slaughtered at one time for tuberculosis than it is to sell 100 sterile cows in groups of three to five during an interval of six months, but the loss to the herd is identical.

In 1909 the 876 animals were handled essentially as a single unit — that is, any animal in any stall in any stable might be transferred to any other stable or stall. There were three milk barns and several barns for the young stock of varying ages. The bull calves were sold at a few days old to persons who grew them for veal. The heifer calves were taken when a few days old to a calf barn, an old structure already existing when the estate was

purchased, without remodeling. There were several rooms in the structure, some with ground and some with board floors. The rooms accommodated anywhere from 20 to 40 or 50 calves each. The calves were generally turned loose, but at milk-feeding time were stanchioned. A paddock was available for exercise.

When the calves were about six months old, depending somewhat upon the crowding, they were removed to one of the heifer stables, where they were detained until pregnant. They were stanchioned in two parallel rows, tail to tail, and fed from a continuous manger. In summer they were placed in pasture.

When safely pregnant four to eight months, according to the degree of crowding, in the respective stables, the pregnant heifers were placed in vacant stanchions in the milk stables, held there until parturition was imminent, and then removed temporarily to maternity stalls.

The three milk barns, A, B and C, are modern structures of a capacity of about 200 cows each. They are spacious, well lighted and ventilated, with cement floors, gutters and mangers. The stanchions are in two parallel rows facing each other. A transverse aisle divides the barn into quadrants of fifty cows each. There are two paddocks with each barn, into which the animals are turned daily for exercise. One-half the cows are turned out at a time. One quadrant goes into each paddock. The paddocks are without food or water. The milk cows are not pastured. Originally the milk barns had continuous mangers for food and water — that is, each quadrant of fifty animals had a common feed and water trough — but this arrangement was later altered in order to control tuberculosis. In one milk stable the stanchion row was cut into single stalls, each with an individual manger and water bucket. In the other two milk stables the stanchions were divided into double stalls with common feeding and watering facilities for the two cows. Separate brooms and stable utensils go with each single or double unit.

The herd as a whole has been divided into large units. The three milk barns constitute each a wholly separate unit. Each unit has:

(1) Its own complement of workmen.

(2) Its group of breeding bulls.

(3) A permanent stanchion for each pregnant or freshly calved heifer entering a given barn, which she does not vacate, except at calving time, until she leaves the herd.

(4) A maternity barn with sufficient box stalls to accommodate the 200 cows in the unit at calving time.

(5) A calf barn with six tight partitioned box stalls designed to accommodate four heifer calves each.

The losses in the herd, aside from the tuberculosis, have occurred chiefly because of abortion and sterility in cows and heifers and calf scours and pneumonia in the heifer calves. Our data upon the various losses are at many points imperfect, but upon the whole are unusually accurate. The data regarding the mortality in new-born calves and the rate of abortion in heifers during first pregnancy are quite complete and accurate.

The data permit of varying groupings in order to bring out some of the most important truths. On May 3, 1909, the total number of females in the herd was 876 animals. From May 3, 1909, to December 31, 1911, inclusive, there were born 491 heifer calves. Added to the original number, these give a total of 1,367 animals. During this period of two years and four months 492 cows were killed or sold, chiefly because of tuberculosis and sterility. Twenty of the heifer calves died of or were killed for tuberculosis before reaching breeding age, and 249 of the heifer calves died from scours, pneumonia, and other causes, leaving out of the total of 491 heifer calves born 222, or 45 per cent, which reached breeding age. On December 25, 1911, the herd totalled 623 as compared with 876 in May, 1909, a deficit of 253 females or a loss of 29 per cent in 32 months.

During this same period there were 123 abortions, so that, of a total of 704 pregnancies, 30 per cent aborted. Assuming that the sexes of the aborted fetuses were essentially equal in number, 106 of the heifer fetuses were aborted. Ninety-nine of these abortions were in heifers pregnant for the first time, so that, out of the entire group of animals, 46 per cent of the total number of abortions occurred during first pregnancy.

In the eight months of 1909 for which we have records, out of 145 heifers pregnant for the first time, 14, or 9.7 per cent, aborted. In 1910 there were 164 heifers in first pregnancy, out of which 48,

or 29.3 per cent, aborted. In 1911, there were 77 first pregnancies, of which 29, or 37.7 per cent aborted. In 1912, there were 56 pregnancies, out of which 35, or 63 per cent, aborted. In 1913, there were 44 heifers in first pregnancy, of which 13, or 29.5 per cent, aborted.

In 1912, owing to the very great discouragement regarding the mortality from white scours and pneumonia in calves and the high rate of abortion in heifers pregnant for the first time, the establishment decided to abandon temporarily the raising of heifers and sold 118 for veal. As a consequence, in the early part of 1914, of those heifers which were left over from 1912 — that is, those which were born in 1912, prior to the sale of the 118 for veal — only three conceived, all of which calved.

In 1910 I had been consulted regarding the diseases interfering with breeding in the herd and had suggested certain measures to reduce the mortality among the young calves. As these measures seemed to the management too complex, they were not put into operation. Consultation was had also with the management of an establishment engaged in the manufacture of sera and other biologic products. Cultures were made from the feces of the diseased calves, and bacterins or sera were compounded which were used in an effort to stem the mortality. These, however, were unavailing. Finally, in 1912, the recommendations made in 1910 were revised, simplified, and put into partial operation.

Early in the history of the herd, the calves were allowed to nurse the mother for a time and no precautions were taken regarding the cleanliness of the dam. When the calf was five or six days old, so that the milk of the cow was marketable, the calf was removed to the calf stable and fed at first upon raw mixed whole milk and later largely upon mixed skimmed milk, which was supposedly pasteurized, but upon investigation was found to be not at all sterile. The process was not reliable. Some tuberculosis followed. The mortality from scours and pneumonia was high. Later the pasteurization was gradually rendered more and more reliable, but apparently did not reach the point of sterilization. However, it was sufficiently complete to check materially the spread of tuberculosis from the milk, without greatly affecting the mortality from scours and pneumonia.

The revised plan for the management of the new-born calf was to take the cow out of the milking stable before calving, give her a thorough bath with soap and warm water, to which a considerable amount of antiseptics was added, and then place her in a well disinfected and freshly bedded maternity stall. She was kept clean, the posterior parts, with the udder, being given special attention, up to the time of calving, and during the period that the calf was allowed to remain with her.

At the age of ten days the calf was removed to the calf stable and placed in a group of from twenty to thirty or more calves of similar age. The cow was then returned to the milking barn. The feeding of the calf after entering the calf barn was not changed materially. It was fed upon pasteurized milk, possibly pasteurized more carefully than is common in dairies.

Under these precautions, which were far from perfect, there was a sensible reduction in the mortality from calf scours and pneumonia (see Chart III), and as a consequence there was a larger number of heifers annually to be bred. These heifers began to calve in October, 1914. Of these pregnancies, the first fifty-five terminated without an abortion. Later, abortions appeared. In the forty months from May, 1909, to August, 1912, inclusive, there were observed amongst the original 876 dairy cows, heifers and heifer calves then on hand, and their progeny of breeding age, 203([1]) abortions, which included presumably 101 female fetuses and 184 heifer calves born during the same period died within a few days after birth, a total of 285 prospective dairy cows, or a prospective loss of 7.1 females per month. Out of the 593 heifer calves born during this period, but 95 calved at two years old, or an average of 2.7 per month, so that the heifers raised did little to replenish the herd. The average deficit in dairy cows was 110 per annum or 9 per month, and 7.1, or 79 per cent of these are at once accounted for by the recorded abortions of heifer fetuses and deaths of heifer calves from scours.

[1] In some of the data those calves which were alive when born, but died almost immediately, were classed as abortions; at other points in the data they had been included under calf scours and pneumonia. These differences in classification cause slight apparent variations in the data, which, however, are not essential.

With the high abortion rate, sterility inevitably played a highly destructive role. Had the herd been maintained simply at 504 and no account made of heifers calving in first pregnancy, there should have terminated during the forty months 1680 pregnancies against a recorded total of 924, or a deficit of 756 pregnancies, or 45 per cent. A large part of this deficit needs be attributed to sterility, but exact figures are not available. It is clear, however, that temporary sterility, resulting in delayed breeding, and permanent sterility, necessitating the sale of the cow, caused enormous losses. The value of growing sound fertile heifers is brought out in sharp contrast when we compare the heifers' calving and the monthly deficits in dairy cows under the two plans of growing calves in herd B. In the first group, with an average of 2.7 calves per month from heifers in first pregnancy, there is a deficit of nine cows per month in the dairy herd. In the second group, since the first heifers began to calve, there has been a monthly average of six calves from heifers in first pregnancy, or more than double the number in the first group.

Chart III brings out in a concrete way the changes in this herd, which we attribute to the feeding of the calves. In the first group of 593 heifer calves born, 184, or 31 per cent, perished within a few days after birth from white scours or pneumonia; in the second group, out of 904 heifer calves born, 203, or 22.4 per cent, succumbed from the same causes during the first few days of life. There follows a very interesting general reduction in the mortality of the calves. Thus, in the first group, by the time they had reached breeding age, 51.4 per cent of the heifers and heifer calves had died; an additional 19.9 per cent had been sold for veal. Thus, only 27.8 per cent of the total number of heifer calves born conceived. In the second group, when the mortality from scours had been definitely reduced by 8.6 per cent, the general reduction in the mortality before breeding age became very marked, so that the deaths up to breeding age, including those animals which would not conceive, totals 30.7 per cent, against 51.4 per cent in the first group. Instead of 27.8 per cent having conceived, as in case of group I, 44.2 per cent conceived. There should be, of course, a deduction made from this of the 19.9 per cent of heifer calves sold for veal in the first group. Had they

CHART III

Calf Scours and Pneumonia, and Abortion and Sterility in Herd B.

Time Covered	Heifer Calves Born		Died of Scours and Pneumonia	Sold as Sterile	Killed account Tuberculosis	Miscellaneous Deaths	Sold for Veal	Conceived	Pregnancy terminated		In Herd Pregnant	In Herd not Bred
									Calved	Aborted		
May 1. 1909 to Aug. 31. 1912. 40 Months	593	No	184	8	56	57	118	170	95	75	0	0
		%	31	1.4	9.4	9.6	19.9	27.8	55.9	44.1	0	0
Sept 1. 1912 to Oct 31. 1916 50 Months	904	No	203	6	13	58	0	382	203	22	157	245
		%	22.4	0.6	1.3	6.4	0	44.2	90.2	9.8	17.4	27

been permitted to live, 16.4 per cent of these would presumably have died from various diseases before they conceived, while in the second group there remain 27 per cent of the heifer calves which have not bred.

The most striking differences between the behavior of the two groups of calves when they have reached breeding age is in the rate of abortion. In the first group, 55.9 per cent calved and 44.1 per cent aborted; in the second group, 90.2 per cent have calved and 9.8 per cent have aborted. We have been unable to find any explanation for this change in the rate of abortion except the method of feeding the heifer calves. It is well recognized that the rate of abortion shifts from year to year in any herd and that sometimes the change is very abrupt and not readily explained. In such cases, however, the abortion rate affects the animals of different ages very much the same. That is, if the abortion rate falls in heifers, it does the same in adults; if it drops materially in adults, it probably, though not so surely, drops also in heifers. In this herd, however, such an explanation is untenable because the change in the abortion rate in adults has not been material. In the period covered by the first group — that is, in the period from May, 1909, to September 1, 1912, inclusive — the abortion rate in adult cows in their third or later pregnancy was 16 per cent. During the period covered by the births of heifer calves in the second group, the abortion rate has been 12.2 per cent — a variation of 3.8 per cent. The reduction in the abortion rate in adult cows also permits of a logical explanation. During the time covered by the second group, the diseased uteri of adults have been systematically douched and disinfected much of the time, which naturally removes much infection and renders pregnancy safer. In the first group the abortion rate during first pregnancy was 28.1 per cent higher than the abortion rate in adults. In the second group, the abortion rate in heifers is 2.3 per cent lower than in the adults. The change was abrupt and corresponded exactly with the change in the feeding of the calves. That is, between the time when the calves grown in the first group had completed their first pregnancies and the first pregnancies of the second group had begun to terminate, the change in the rate of abortion shifted suddenly and the relationship has since remained constant.

Examining the data in detail with reference to the observed abortions during first pregnancy, we find that in 1909 and 1910 there was a larger number of pregnant heifers than is reported for 1911 to March, 1914, inclusive. We have no data regarding the mortality in the heifer calves which were pregnant for the first time in 1909 and 1910. It is highly suggestive, however, that there were 145 terminations of pregnancy in heifers in the last eight months of 1909, or an average of 18 for each month, and during this period the abortion rate was 9.7 per cent. In 1910 there were 164 heifers pregnant for the first time, an average of 14 per month, or 22.2 per cent, less pregnant heifers per month than in the previous year, and the abortion rate advances to 29.3 per cent. In the next year there is a marked decrease in the number of pregnant heifers from 164 to 77, or a decrease of 53 per cent in the number of first pregnancies, and the abortion rate advances to 38.7 per cent. In the following year there was a further drop to 56 first pregnancies, or a decrease of 27 per cent in the number, and the rate of abortion advanced to 63 per cent. In the next year there was a decrease in the number of pregnancies to 44, or 20 per cent, with a decrease in the abortion rate to 29.5 per cent.

The actual differences in the efficiency of the two groups of heifers is not told by the chart. I have insisted that the disease must be measured by the volume of sterility, abortion, premature birth, and metritis with or without retained fetal membranes, and more recently in a large measure the health of the new-born calves. While they do not appear in Chart III, the variations in the other phenomena are parallel to those of abortion. It is the practice to breed heifers at fifteen months in order to have them calve at two years. In the first group, where abortion averaged 44.1 per cent, first pregnancies terminated upon an average at twenty-eight months. In the second group, with an abortion rate of 9.8 per cent, the first pregnancies terminated when the heifers had reached an average age of twenty-five months, or three months earlier than in the first group. In the first group, the abortions and the inevitably associated premature births shorten materially the average age of the heifer when her first pregnancy terminated, so that there was an even greater variation in the

average age at which the heifers calved than is indicated by the data. In Chart III it is shown that permanent sterility demanding the sale of the heifers for beef caused appreciable losses, while in the second group these losses become insignificant. In each group, however, the permanent sterility is insignificant from an economic viewpoint as compared with the temporary sterility. The 203 heifers in the second group in Chart III, calving on an average three months earlier than those in the first group, rendered a total dairy service of 609 months before the beginning of dairy service in the first group. If ten months are accepted as a dairy year (allowing two months of rest before calving), the increase of efficiency equals the dairying efficiency of 61 cows for one year — a very important economic item.

It is important to realize that the temporary sterility is largely embryonic abortion or other equally significant disease of the genital organs referable to the abortion infection. The breeder should realize fully that, with some exceptions, temporary sterility is not only contagious abortion, but that in a large proportion of such cases conception has occurred, but the embryo is expelled ere it has reached dimensions to attract attention. This is brought out in Chart II, where numbers 34, 34a and 34b have been served repeatedly with notable skips in estrum and a very high rate of known abortion (50 per cent), while numbers 49 and 49a have bred far more promptly with but a single skip (49b in second breeding) without an abortion thus far.

The changes in the prevalence of metritis and retained afterbirth in Herd B have been equally conspicuous. In the first group, retained fetal membranes were common, not rarely proving fatal, frequently destroying the dairying efficiency, and often causing incurable sterility; in the second group, metritis and retained afterbirth have vanished as an important economic factor.

With these changes comes an inevitable increase in the dairying efficiency of two-year-old heifers. Whenever abortion is rampant in heifers (or, for that matter, in cows of any age) the dairy efficiency of those calving at full term is inevitable greatly lowered. As the ratio of abortion increases, the dairy efficiency of those which do not abort decreases. That is, the higher the rate of abortion in a group of heifers in first pregnancy, the higher the

virulence of the infection in the remnant of the group which calve at full term, and this intensity of infection affects the efficiency of lactation. Before the real significance of contagious abortion can be comprehended, the entire complex group of disasters which the disease causes must be recognized as a unit — as a single disease.

In Herd B tuberculosis and the sterility-abortion-calf scours group has each played its part. While measures have been instituted somewhat independently for the suppression of each, they have proven complementary at every point, and the two diseases have been simultaneously repressed. The herd had shown an annual deficit of several thousands of dollars; the past year has shown a profit. While this economic revolution is attributable in a large measure to the better control of tuberculosis, it is in much larger measure due to the repression of the sterility-abortion-calf scours group. Excluding the control of tuberculosis by quarantine or by slaughter, any sanitary measures intelligently applied for the suppression of one will favorably affect the other disease.

The large number of heifers in first pregnancy during 1909 and 1910 would suggest that the mortality in the group when new-born calves was low. The only exception to the parallelism between the number of heifers which were pregnant for the first time and the inverse ratio of abortion was that of 1913. In a general way the variations in the rate of abortion in first pregnancy have been remarkably in harmony with the variations in the mortality in the group as young calves from calf scours and pneumonia.

The chart shows apparently an undue variation in the abortion rate of the two respective groups when compared with the variation in mortality from white scours and pneumonia. This, however, seems to admit of rational explanation. The calf scours and pneumonia, so far as we understand the problem at present, are due to a mixed infection. The influence of the infection upon the future breeding of the calf in case it lives may be dependent more definitely upon one infecting agent than upon another. Abortion itself, as understood, is by no means always a pure infection, but is generally mixed. That is, we are inclined to the belief that, although the abortion bacillus is the fundamental cause of abortion, it is aided materially by the presence of secondary

invaders, such as various members of the colon group and streptococci. So also are scours and pneumonia of calves. Some cases are unquestionably due to the Bang abortion bacillus alone, or practically alone, but in the vast majority of cases the investigator observes, not the abortion bacillus, but the colon or streptococcus. It is quite possible, if not probable, that the abortion organism is commonly present and plays its part, but we cannot know this with our present methods of search, because the secondary invaders are so profuse in their growth that the abortion bacillus, if present, is veiled and exceedingly difficult of recognition. What appears a probable explanation for the greater variation in the abortion rate than in the mortality from white scours in the two groups is that, in pasteurizing the milk for the second group, the abortion bacilli were very largely destroyed without the colon bacillus being so completely eliminated. It is the common report in the pasteurization of milk that the colon group of bacilli are among the last, if not the last, to be destroyed by heat. It is known that in this herd the pasteurization of the milk did not mean sterilization. The milk had to be fed soon after its pasteurization or it decomposed and acquired a very foul odor, due presumably to the multiplication of the colon organisms in the absence of the restraining lactic acid bacilli.

It is not, however, entirely a question of the preparation of the milk which fixed the frequency and intensity of the white scours. Running together in a common room, any sound calves were always exposed to any infection which a diseased one might introduce. If a new calf, already infected, were brought in from its mother, the pasteurization of the milk could not prevent the spread of the infection to the other calves. The plan of growing calves in common rooms could not prevent the spread of scours, and the success could only be of that partial character which the records bring out so strongly. At any time, upon the introduction of virulent infection by means of a diseased calf, the infection may very readily break loose as a storm. This is quite well illustrated in the calf records of the herd from December 1, 1915, to February 1, 1916, inclusive. During this period there were born 69 calves, of which 34, or 50 per cent, died. The management then made a change by which the new-born calves were held in clean stalls in

isolated groups of four and greater care was taken to prevent the spread of infection. From February 1 to October 22, 1916, inclusive, there were born 153 calves, with a mortality of 7 calves, or 4.6 per cent. The change in the mortality must, therefore, not be attributed entirely to the preparation of the milk, but involves other elements in the care as well, and especially involves the very important element of contagion from calf to calf.

Various theories might be offered for the differences in the rate of abortion in these two groups of calves, but no one of them, so far as we can find, except the change in the feeding of the calves, will withstand a critical study. It is quite generally believed that the disease spreads rapidly by ordinary cohabitation. The calves of the two groups, however, were kept in the same stable. In the heifer stable, where the heifers are bred for the first time, a considerable number of the first group still remained when the second group began coming in. Naturally, they were bred to the same bulls. Each group of heifers, after they had conceived and were four to six months pregnant, were taken, according to convenience, to one of the three dairy stables and placed in any vacant stanchions which might exist. They were subjected constantly to the same cohabitory exposure; the two groups were cared for by the same group of men, of constantly shifting personnel; they occupied the same stables, paddocks, and pastures; they used the same water, and they were fed upon food of the same character from like sources.

If there was any material change in the character of the bulls used, so far as known, it consisted of the use upon the second group of some young bulls which had been grown in the group as part of it, and consequently referred back to the same cause — the method of feeding the new-born calf.

It seems impossible, after studying the records of the herd very carefully, to find any explanation for the change in the abortion rate during first pregnancy except the feeding of the new-born calves. The feeding and care of the new-born calves has been anything but perfect, even under the amended plan. What the results would be were the mortality from white scours and pneumonia practically eliminated, as we believe can be readily done, we do not know.

A very interesting element in the data is that the calves, as they were grown, were each exposed to certain important sources of infection. In the first place, all the calves in both groups were equally exposed to any intra-uterine infection. We know that this is an important element in the production of scours. In the second place, all the calves in both groups were exposed alike to the contamination of the milk within the udder. It has been shown recently that the bacillus abortus exists commonly in the udders of dairy cows. Schroeder contends that the udder is the principal reservoir for the multiplication and the indefinite persistence of the organism in the body of the cow. Certainly at present it is the most frequent known source from which the bacillus may be obtained. The exposure of the two groups of calves to this source of infection has been alike for the first ten days of the life of the calf. When the calf is taken from the cow and placed upon pasteurized milk, the exposure of the two groups is alike except for probable differences in the process of pasteurization. There is every reason to believe that the process has been much more carefully carried out for the second group. Recently the milk has been boiled after the first ten days, but the heifer calves affected by this change have not yet reached breeding age.

The great difference in exposure to the infection of contagious abortion and to the colon and other bacilli has been in the care of the cow at the time of calving and immediately afterwards. Previously it was supposed that any cow was clean enough to give birth to a calf and that, after she had given borth to it, she was clean enough for the calf to suck. In large herds such as Herd B there is inevitably a high degree of virulent infection in the genital tract of many of the cows. Before and after calving, large volumes of virulent discharges loaded with the abortion bacilli, colon organisms, and streptoccocci, flow down the tail and thighs, to soil the exterior of the udder and teats. If the cow is not washed and her udder is not kept carefully cleaned, the calf inevitably takes with its milk a large volume of virulent organisms which have reached the exterior of the udder from the genital tract. Especially is this true when there is marked metritis, with or without retained afterbirth, so that pints or quarts of virulent discharges are daily making their way down along the thighs

and tail, to contaminate disgustingly the udder and teats. The evidence in Herd B goes to show that this is one of the most dangerous sources of infection. It appears, so far as the danger to calves is concerned, to be infinitely more important than the infection within the udder. Neither do we know precisely what influence the infection upon the exterior of the udder and teats exerts upon the degree of infection within the udder. Schroeder contends, with apparent reason, that the infection in the udder may be deposited there from the blood stream, but he has also shown that it may be injected into the udder and believes that it may gain the udder by being carried from the diseased to the healthy gland upon the hands of the milker. If it can be carried readily by the hands of the milker, certainly such virulent discharges as are often observed flowing down from the vulva would have a much greater danger for the udder than the milker's hands. This is in complete conformity with clinical observation. In Herd B during the growth of the calves under the first plan, mammitis, or garget, was rampant, and many cows had to be discarded because of such disease. Under the second plan, the frequency of mammitis has been greatly lessened.

THE INFLUENCE OF COPULATION AND OTHER AGENCIES UPON THE AGGLUTINATING POWER OF THE BLOOD

In the researches of this department, it has been observed for several years that the results of the agglutination test of the serum of cows upon emulsions of abortion bacilli varied greatly at different times, but no definite study was undertaken to determine the explanation or reason for those variations. In young animals especially, the results have proven highly erratic and confusing.

It was early observed that the blood serum of new-born calves usually, but by no means always, tested negative at 1–10. Occasionally it caused an agglutination in calves at 1–100 at the time of birth. The serum of those calves testing negative at 1–10 at birth quickly separated into two groups. In the first group, chiefly in herds where the abortion rate is low, where there is little metritis in the cows with or without retained afterbirth, and where the calves do not suffer prominently from scours, the agglutinating power of the blood of calves remains weak or nil at 1–10 for

two, three, or more months, then becomes positive at 1–10 and tends to remain almost static, advancing occasionally to 1–25 up to sexual maturity. If a calf born in a highly infected herd, but whose blood reacts negative at 1–10 at the time of birth, be fed upon boiled milk, the agglutinating power of the blood tends to remain feeble or negative at 1–10 for several months and then advance gradually up to puberty. If a calf born in a highly infected herd, although its blood is at first negative at 1–10, is fed raw milk from the herd, the agglutinating power of the blood advances rapidly and at the age of seven to fifteen days may acquire an agglutinating power of 1–50, 1–100, or even 1–1000. The high agglutinating power persists for a few weeks, then slowly recedes, and at ninety to 100 days may be almost, though not quite as feeble as in those calves in herds suffering only slightly from the abortion group of diseases or those calves fed wholly upon boiled milk.

The reaction then remains variable until the calf, of either sex, reaches sexual maturity. Some calves then show a sudden moderate increase in agglutinating power, in such a manner as to suggest that the advent of sexual life may or does tend to cause an increase in the agglutinating power of the blood.

When sexual desire is permitted to result in copulation, a sudden and marked advance in the agglutinating power of the blood of both sexes occurs so frequently that it appears that sexual activity definitely and generally intensifies the reaction. In the researches of this department, the opportunities for studying this point have been greatly limited, so that our data are restricted in volume and weakened by the infrequency of the tests.

The data at hand relating to copulation, though small in volume and admittedly very fragmentary, appear to be worthy of record in order that the attention of investigators may be directed thereto. The influence upon the agglutinating power of the blood appears to be most marked from the first copulation, although repeated copulation appears for a time to advance the agglutinating index.

Some of these observations have already been recorded in the Annual Report for 1913–1914, the chart accompanying which is reinserted here as Chart IV. This, with Chart V, indicates the variations in agglutinating power in response to the influences

named. The findings here recorded justify further studies because, if it is found that sexual maturity and copulation intensify the agglutinating power of the blood in both sexes, it would provide valuable suggestions regarding the nature of the infection and some of the principles involved in its control.

CHART IV

AGGLUTINATION · EXPERIMENTS · ON · HEIFER · CALVES
AFFECTED · WITH · DIARRHEA · AND · AT · TIME · OF · BREEDING

NUMBER	DOB.N	WHITE SCOURS	BRAN VEN SCOURS	MAXIMUM AGGLUTINATION BEFORE BREEDING DAYS OLD	REACTION	DAYS OLD	REACTION	BREEDING DATE	MAXIMUM AGGLUTINATION AFTER BREEDING DAYS AFTER	REACTION	DAYS AFTER	REACTION	RESULT.
875	11-26-12	-	+	130	10	357	-	1-7-14	2	300	28	50	A 8-12-14
880	12-18-12	+	+	108	600	335	-	1-24-14	7	1000	33	600	A 8-12-14
874	11-19-12	-	S	137	10	364	10	417	-	1-5-14 1-25-14	4	300	30	300	A 6-19-14
881	9-18-12	+	S	193	-	426	25			1-4-14	5	1000	31	50	A 9-11-14
866	12 16 12	-	S	110	10	357	10	390	-	1-14-14	21	50			A 6-28-14
876	12-8 12	-	S	118	-	345	10	398	50	1-10-14 1-29-14	2.5	50			A 9-8-14
870	9-6-12	-	+	211	25	438	-			1-6-14 1-27-14	3	1000	29	-	A 8-31-14
863	12-24-12	+	-	102	-	329	-	382	50	408	50	2-9-14					A 9-3-14
882	9-15-12	-	S	204	-	431	-	484	600			1-21-14	15	50			A 8-24-14
865	9 16 12	-	S	201	100	428	-					12-31-13 1-18-14	9	50	35	10	S. TB. REACTER
861	9-12-12	-	S	205	10	432	-					1-18-14 2-13-14	11	-	37	600	P 11 9-14
883	12 28 12	-	+	98	-	325	-	378	25	404	10	3-30-14 5-10-14					P 11-13-14-TB
877	12-9-12	-	+	117	-	344	10	397	30	423	30						
879	1 13 13	-	+	80	10	307	-	360	300	386	25						
884	9 16 12	-	+	211	25	438	-	491	-	517	10						
868	5 28 13	+	S	8	-												
878	2 22 13	+	S	82	-												
873	2-28-13	-	S	36	-												
872	3 8 13	-	-	28	10												
869	2 2 13	-	S	62	-												
	2-7-13	-	D	57	-	284	-										
	12-6-12	+	D	120	10												
	3 23 13	+	D	13	600												
	1-24-13	+	D	71	10												
	2-17-13	+	D	47	200												
	7-13-12	-	D	266	50												

CHART V

Research upon Animals Numbers 10, 7, 11, 50, 1, 3 and 101.

Number 10 is a male; the others females.
Numbers 1, 3, 7, 10 and 11 were grown upon boiled milk.
Numbers 50 and 101 were grown upon raw milk.
The spaces between the regular perpendicular lines represent calendar months and the dotted perpendicular lines between the former represent copulations. The angular tracings in solid black lines indicate complete agglutinating power up to the horizontal line it reaches, and any extension in the form of dotted lines indicates that the agglutination was partial only.

No. 10

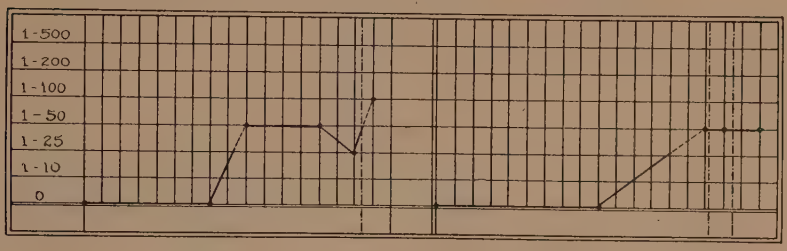


Fig. 1

No. 7

Fig. 2

No. 11

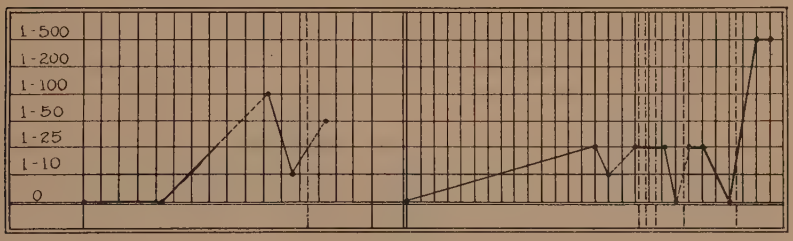


Fig. 3

No. 50

Fig. 4

No. 1

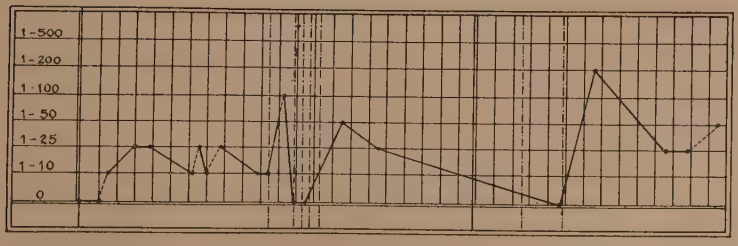


Fig. 5

CHART V — (*Continued*)

No. 3

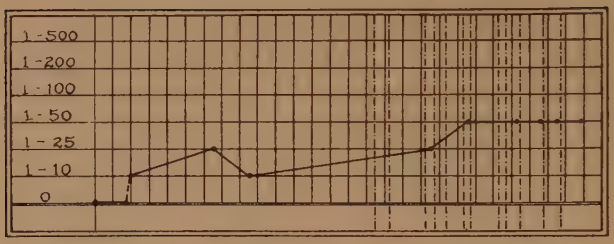


Fig. 6

No. 101

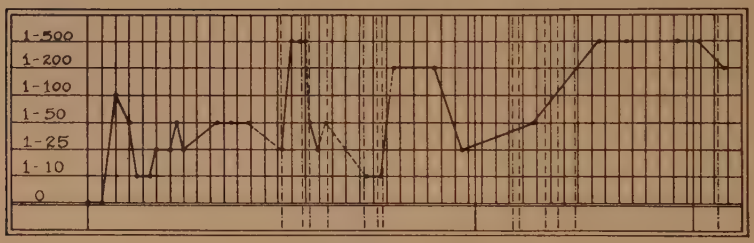


Fig. 7

DISEASES OF NEW-BORN CALVES

The diseases of new-born calves constitute a highly important part of the problem of the diseases of breeding cattle. This group presents three important considerations:

(1) The diseases of the individual new-born calf as a peril to its life, vigor, and growth.

(2) The sick calf as a menace to the health and lives of calves with which it may come in direct or indirect contact.

(3) The influence of the diseases of the new-born calf upon its efficiency as a breeding and dairy animal in case it survives and reaches adult life.

The two outstanding diseases or aspects of the group are calf scours, or white scours, and calf pneumonia. Associated with these, and inseparable as yet bacteriologically and clinically, is the

acute septicaemia of new-born calves and the acute arthritis, or joint ill. Other pathological conditions arise more rarely belonging to, or at least inseparable from, this great group.

The diseases of new-born calves have received very scant attention, both bacteriologically and clinically. One of the best known studies upon this question is that by Nocard in 1901, at the close of which he announced that the infection of white scours invades the body of the calf chiefly or wholly through the broken umbilic cord. These studies of Nocard were not extensive and have not been seriously followed by other investigators. His conclusions, based upon inadequate observations, have led to much confusion in the handling of the disease. Almost no accurate observations upon this subject have been recorded anywhere. Reliable investigations are difficult to make. Breeders of pedigreed cattle do not care to have research work upon the calves in their herds because of the almost unavoidable publicity, which they abhor, and because of the value of the calves, which causes them to be exceedingly fearful of possible losses due to the research work. The ordinary dairyman, who does not care particularly about growing his calves, because they are of comparatively low value, does not facilitate research work because he considers it not worth the trouble. For these and other reasons, this group of diseases has gone without any very extensive or accurate observations. The handling of this group has been left very largly to the manufacturers of biologic products or the compounders of various drugs. These manufacturers send out their drugs or biologic products and receive from users of them favorable reports in some cases, which they very naturally use as advertisements for their preparations. Such reports have little value. They are generally very misleading because the cessation of the scours or pneumonia is recorded and the failure of calves to recover usually goes unrecorded. The result is a badly prejudiced report, and the conclusions based thereon are unavoidably erroneous.

Informal clinical observations have been made for a number of years. It is observed that calf scours and pneumonia, like abortion, sterility, and retained afterbirth, are exceedingly erratic in their appearance and disappearance from a herd. No breeder can well know just when an outbreak is to occur or how serious

it may prove. Sometimes a single calf sickens and dies, and apparently that is the end of it. In other cases the disease breaks as a storm in a stable and kills 50 to 100 per cent of the new-born calves over a long period. In large establishments, like Herd B, q. v., the average mortality from white scours and pneumonia is very high, usually reaching 25 to 30 per cent or more. Since in Herd B the calves are of moderate value only, it might erroneously be thought that the management was indifferent and that energetic measures for the control of the disease were consequently wanting. In the data hereto attached, we are dealing with a herd of an entirely different type, where the new-born calves have an estimated value of from $100 to $1,000 each, with an average estimated value of probably $250, so that there is ample reason for doing all that can well be done to reduce the mortality from this group of diseases. In spite of the great value of the calves, out of the subjoined list (Table A) of ninety-eight calves that were born from January 1, 1915, to June 1, 1916, at the time of the beginning of our experiments, forty calves — 41 per cent of the total — died from scours or later from other diseases belonging to this group and following as a consequence of this infection. It is consequently not a disease of cheap animals. If vital statistics could be had in the highly valuable herds of the country, it would be found that the losses from this group of infections are really quite appalling.

The group is exceedingly complex and the outstanding members of it have been regarded generally as separate diseases. Breeders speak of calf scours, of calf pneumonia, of arthritis (joint ill), and of calf septicaemia. When these various aspects of the disease are carefully studied bacteriologically and clinically, no material differences can be distinguished. Inevitably there is a mixed infection and it is impossible to place the blame for the entire group upon any one disease-producing organism. It is found, however, upon examination, that there stand out prominently two groups of organisms — the abortion bacillus of Bang and the colon group, mixed with which there is a variable number of other organisms existing very commonly and capable of inducing disease when gaining entrance into the tissues of the body. So far as we know, the organisms which cause this group of diseases in calves exist always in the organs of adult cattle. The

injuries which they are capable of accomplishing in calves must be referred to the two facts that the calves are unaccustomed to these organisms — that is, they have acquired no working immunity against them — and that the volume and virulence of the organisms which gain admission to the body of the calf are unduly great, thus breaking down such feeble barriers as nature has at the time provided.

There are three principal sources of these infections, from which the calf may obtain them in large volume and of virulent type. In the first place, the calf may and does obtain these during intra-uterine life from the infected uterine cavity of the mother. This source has been quite clearly established in the course of the investigations which we have made. The infection of the fetus is through the chorion from the utero-chorionic space. In those cases where the abortion infection is severe in the uterine cavity of the pregnant animal, the uterine seal is frequently broken down so that any infection which may exist in the vagina may and does readily extend into the uterine cavity, bringing about there a mixed infection, consisting of the abortion bacillus, which, according to our present knowledge, is the original offender, colon bacilli, and other organisms. Eventually, these grow through the chorion and gain thereby the amniotic or the allantoic fluid. If they gain the allantoic fluid, they may possibly but not at all readily enter the fetal body through the urachus, and urinary bladder. Far more readily and probably they may pass from the allantoic fluid through the thin intervening membrane into the amniotic fluid.

Whether the organism reaches the amniotic fluid directly through the chorion or indirectly from the allantoic fluid, is not material. The important point is that the infection may and does penetrate to the amniotic cavity and is swallowed by the fetus. The amniotic fluid which is swallowed by the fetus does not pass through the anus, but is absorbed from the intestines and enters the fetal circulation. Thus, the intestinal walls constitute a filter for the swallowed fluid. The fetus is constantly shedding hairs, as soon as it has any hairs to shed. It also sheds epithelium. The hairs, epithelium and other solids suspended in the amniotic fluid are filtered out after being swallowed and remain in the posterior portion of the alimentary tract, to contribute to the formation of

the meconium. Any bacteria which gain entrance to the amniotic fluid are likewise swallowed and are as a rule detained in the large intestine as an integral part of the meconium. The most marked other addition to the meconium is that of the bile from the liver. This becomes desiccated and, mixed with the fetal hairs and other debris, forms in compact pellets or masses, which we know as the meconium. The biliary salts, entering into the meconium, serve to restrain bacterial growth, and it is only when the volume and virluence of the bacteria swallowed reach a preponderating intensity that the restraining power of the inspissated bile is overcome and disaster to the fetus follows. So long as the biliary salts are competent to hold in leash the bacilli which are swallowed by the fetus, the amniotic fluid remains clear, while the allantoic fluid, which is not subject to this filtering process, is very frequently clouded. If, however, the infection swallowed by the fetus is sufficiently virulent to overcome the barrier afforded by the bile in the meconium and by the intestinal walls themselves, disease of the fetus at once supervenes. Severe fetal diarrhea then becomes established; the fetus has white, or calf scours. The intestinal contents are then emptied into the amniotic cavity, so that when the calf is born or is expelled dead a great mass of diarrheic feces is found distending the amniotic sac. We have observed as much as five to ten gallons of such feces expelled from the amniotic sac prior to and during the expulsion of the fetus. In such cases the exterior of the fetus is dirty, covered over with soft, yellow feces. Very frequently the fetus is dead at the time of expulsion. Sometimes the fetus is expelled alive and quickly dies. In other cases the calf may recover promptly and grow vigorously.

Most frequently the meconium holds the infection which is deposited therein in abeyance, until the calf is born. Then, with the taking of milk into the alimentary tract, a new opportunity is afforded for the multiplication of the organisms present, and scours more or less promptly supervenes.

The new-born calf is not always afflicted with scours, as revealed by the expulsion of large amounts of thin feces. Instead, it is not at all rare for the new-born calf to be exceedingly listless and dull. It is too weak to get up, and perhaps too weak to stand if it is helped to its feet. During this time, perhaps, it has no evacuation of the bowels. The calf dies from an acute sepsis. This type of

disease is especially common in premature births. Inevitably, a prematurely born calf is highly infected, not with one, as a rule, but with several kinds of bacteria. In many cases the whole system of the new-born calf is overwhelmed. It may be able to stand, but does not eat. Frequently it has a peculiar voice. It bleats continuously, making a sound much like that of a kid or a lamb. In these cases of acute sepsis, the calf dies without the intestines reacting and attempting to throw off the infection by means of profuse intestinal discharges.

If the calf escapes the acute sepsis and survives for a few hours to several days, the infection already in its intestinal tract accumulates force, and calf scours or dysentery becomes established. The symptoms of the scours are necessarily greatly varied. It is frequently known as white scours, but the feces may be white or almost any other color. The first symptoms of approaching scours are vague, and frequently misleading. If the feces from the calf are observed closely, one of the first symptoms seen is some patches of blood covering the fecal pellets. Sometimes there is a cubic centimeter or more of blood spread out over a fecal mass. The blood looks fresh, as if the bowel had in some way been torn and a recent hemorrhage had occurred within the intestine and clotted there. Sometimes the feces are merely flecked with blood.

We do not know how often the feces are bloody prior to the advent of the dysentery. Close observations upon this point are wanting. In those calves which have been watched closely and continuously, however, this premonitory symptom is very prominent, and the question arises: How nearly constant may it be? It is a misleading symptom until one is initiated into the behavior of the disease. The calf at this time appears quite well, and it may be two to four hours afterward before the storm breaks and the calf is observed to be seriously ill.

Another symptom which frequently arises suddenly and indicates the near approach of the storm is arthritis, usually of the stifle or tarsus. At one time the calf is observed playing and perhaps takes its milk with avidity and seems in all respects perfectly well. An hour afterward, it is excessively lame and will bear no weight upon one of its limbs. It is dull and prefers to lie down all the time. If forced to get up, it goes limping about. If the limb is carefully examined, one or more of the articulations will be

found somewhat swollen, an increase of synovia present, and the joint intensely painful upon palpation. The calf breathes somewhat hurriedly and its temperature may suddenly mount to 104 F. or higher. It may still take milk when offered, or may refuse it entirely.

A little later, the dysentery appears in a virulent form. The feces are usually light colored and very tenacious, sticking to the tail and buttocks. The consistency of the feces varies greatly. They may be simply watery discharges and the calf may show considerable tenesmus in evacuating the bowels. The stools are filled with gas bubbles and give off a very fetid odor. Depending upon the intensity of the disease and the vigor with which it is handled, the destiny of the calf varies widely. In many cases it droops and dies within six to twelve hours after first observed ailing, or the disease may be slow, destroying the life of the patient at almost any time up to several weeks.

If, at the beginning of the scours, we destroy the animal, if the new-born calf has died from acute sepsis, or if the fetus has died within the uterus (abortion), an autopsy generally reveals the lesions of acute sepsis; the liver and spleen are dotted over with hemorrhagic spots; the joints are very frequently distended with a dirty flocculent exudate, usually watery, perhaps containing clots of synovia; the joint contents are frequently flecked with blood. The changes in the alimentary canal are usually very slight in the anterior part. The stomach in many cases contains normally curded milk, as if digestion had begun in an orderly manner. Then, as the alimentary tract is followed backward, increasing evidences of disease appear. First, there are here and there small patches of inflammation of the mucosa and as the posterior end of the gut is approached the mucosa is found uniformly highly inflamed, swollen, and intensely red.

This group of cases, in which the foundation for scours has been laid during intra-uterine life and in which the scours may be present at the time of birth, constitutes the direct link in the chain existing between the scours and pneumonia of the calf and the contagious abortion. If a cow has metritis during her pregnancy — the metritis of contagious abortion — which has not been sufficiently intense to destroy the life of the fetus, the meconium of the new-born calf frequently shows culturally the same bacteria as

the uterine cavity of the mother. This is especially marked in those cases of the metritis of contagious abortion complicated by retention of the fetal membranes. Our colleague, Dr. Hagan, removing a cotyledon from such an animal, obtained, deep within the placental mass, where accidental infection was excluded, cultures apparently identical with those obtained from the meconium of the new-born calf. Other data and clinical observations confirm these findings. The disease then becomes, in the strictest etymological sense, hereditary. That is, it is a disease transmitted directly from parent to offspring in utero.

Passing from this group of cases, the new-born calf, if born sound and free from infection, may quickly be exposed to virulent infections from various sources. First of all, and of greatest importance, as indicated by the data in Herd B, are the virulent discharges from the diseased uterus of the cow. It has from the first been fully recognized that the largest volume and most virulent type of abortion infection exists in the cavity of the uterus. This we know is expelled very largely before the calf is born, and an immense volume is thrown out during the birth act. After birth the metritis persisting causes a voluminous discharge of highly infective material which escapes from the vulva and flows down along the thighs and tail to the udder and teats. Unless unusual precautions are taken, the new-born calf takes the infection into its digestive tract with its first mouthful of milk, no matter whether the calf is allowed to suck or whether a careless milker draws the milk for the calf in such a way as to include the virulent discharges. The result is the same. Not only does the calf get in its food in this way the abortion bacillus, but the entire complex infection which has existed in the uterus of the pregnant cow. Especially when there are retained fetal membranes, the infection in the genital discharges is highly complex, revealing largely the colon group which takes so prominent a part in the biology of calf scours.

Even if the udder be washed clean, the interior of the gland itself, as has been shown by Schroeder and others, contains the abortion bacilli. Other disease-producing organisms also exist within the udder to a greater or less extent, especially in the presence of mammitis, so that, whenever the udder is seriously infected, the peril to the calf is great. We know only too well also

that dairymen frequently take the milk from such diseased cows to feed to the calves, as a matter of supposed economy, however false the economy may be.

In our clinical observations, there is constant harmony between the degree of disease in the uterus of the dam and the tendency of the calf to suffer from scours, so long as the scours is primary; that is, so long as the scours infection is transmitted from the mother to her fetus or new-born calf.

A single primary case of intense scours may serve as a leaven for an exceedingly disastrous outbreak in a herd. Veterinarians and breeders have too long been content with the conclusion of Nocard, that the one great cause of white scours in calves is umbilic infection. Time and again we observe where one calf with scours is placed with other calves several days old and apparently well, that the others very promptly contract the disease and suffer very seriously, if not fatally. It has now been very clearly established that the fetus in utero gets calf scours by swallowing the infection in its amniotic fluid. It is equally clear, from the data in Herd B (Chart III), that the new-born calf most frequently gets the infection by swallowing it with its milk. The evidence is so clear and emphatic that it can not be reasonably doubted. Indeed, one should not expect otherwise than that an infectious disease of the digestive tract would be most readily conveyed through the mouth. One sick calf placed in a stall with healthy calves or — which is equally bad from a sanitary standpoint — a sick calf placed in a stall adjoining another stall in which sound calves are kept, with partitions consisting simply of bars or of woven wire, gives every opportunity for the infection to spread from calf to calf. When the sick calf is placed in a stall with well calves, the feces — or, in case of pneumonia, the sputum — is thrown about the stall indiscriminately, the other calves become soiled and lick themselves or the soiled, diseased calf; they suck each other, or pick up the infection from the soiled bedding or food, and swallow a large amount of highly virulent matter.

The crowding of new-born calves together in a single stall, regardless of health or disease, and the building of calf stalls with open partitions are crimes against animal husbandry. The plan is directly opposed to all sanitary considerations and in the present state of the health of our herds is one of the most disastrous prac-

tices in which a breeder or dairyman can indulge. Calves should always have separate stalls with solid partitions so that they are wholly isolated from other calves until their health is assured.

The conclusion of Nocard that white scours is transmitted chiefly through the umbilic wound admittedly contains an important element of truth. There can be no doubt that the infection may thus be spread, but this avenue does not constitute the only way and it is equally certain that it is not the chief avenue of infection. As a matter of course, the navel stump should always be properly disinfected and cared for. That is good animal husbandry and avoids many serious losses, but it is fatal to depend upon this as the cornerstone in the control of such an infection as white scours and pneumonia.

The pneumonia of calves is indistinguishable at present, so far as its bacteriology goes, from calf scours. It attacks ordinarily the older calves. The infection acts very slowly. Either the volume or the virulence of the infection has been less or the resisting power of the calf greater, and the clinical evidences of the disease are delayed in a large measure. However, pneumonia rarely, if ever, occurs without scours. It occurs so frequently, however, without severe scours that many observers fail to note the latter. In fact, digestive disturbances are so common in calves, especially in large dairying and breeding herds, that only a few of the worst cases of scours are observed. In a large proportion of the herds of cattle, there is a mild chronic scours. The feces are fetid and somewhat soft. The bowels are irregular. Sometimes the calf is constipated. All the time, however, the feces are exceedingly sticky and gummy, so that in defecating they adhere to the tail and thighs. It is not uncommon to observe every calf in the herd thus soiled with feces, while the owner believes that his calves are well and absolutely free from scours. The calves eat fairly well and grow some, but the soiling of the posterior parts continues. On the contrary, a calf which is sound never becomes soiled with feces about its tail and buttocks. In defecating the feces never adhere to the hairs, but are expelled free and the calf remains clean.

Closely associated with the scours and pneumonia and constituting a part of the general infection, so far as can be determined at present, is the "joint ill" or arthritis. It is sometimes acute

and sometimes chronic. We have already discussed the acute type. The chronic form assumes usually a rachitic type. The joints become enlarged and hard, and the calf is stiff and refuses to play. There may be a very distinct lameness in one, two or three limbs, but in a general way there is a stiffness of the animal, with inability to move freely and a very marked desire to be let alone and keep still. The calf is very unthrifty and along with the rachitic symptoms there are nearly always evidences of bronchial or pulmonary irritation, the calf coughing frequently. At the same time, the tail and buttocks are soiled with the gummy feces; the general condition is very bad; the calf grows little or none in spite of liberal feeding; after lingering for several weeks, it very frequently dies, though in some cases it recovers slowly and may eventually acquire considerable size and the traces of the severe illness may largely disappear.

There are frequently observed in new-born calves along with this complex infection, abscesses in the lips and cheeks. These tend to interfere very seriously with the welfare of the calf. They rupture into the mouth, and the highly infected pus is very largely or wholly swallowed to intensify the infection in the alimentary canal. The cavity of the abscess ulcerates and becomes exceedingly inflamed and tender. Not infrequently, these abscesses and ulcers seem to play a leading part in destroying the life of the calf. In the midst of the general severe infection, there is very little power in the tissues to heal such ulcers, so that as a general rule they do very badly unless vigorous measures are undertaken to improve the general health of the calf and bring under control the complex general infection, which at the same time is involving the entire digestive tract and the lungs.

Not all calves suffering from this mixed infection show any such severe symptoms as those which we have described. It is an almost universal rule that, soon after a calf is born in a large dairy or breeding establishment, it loses the primitive lustre of its coat, the hair becomes rough and lustreless, and the calf becomes gaunt or pot-bellied. It plays some, but not as vigorously as it should. It eats fairly well, but its appetite is somewhat fastidious. Its growth is somewhat slow. Its feces are pasty and adhere to the tail and buttocks. When the calf reaches twelve to twenty weeks of age and begins to live very largely upon vegetable foods, the

lustre of its coat gradually reappears, the gaunt or pot-bellied condition improves, the calf shows better life, and its general growth and vigor become quite changed. The calf has passed through a long and severe exposure to a mixed and harmful infection and finally, when the highly infected food is largely displaced by hay and grain or grass, it at last gains mastery over the chronic infection and resumes an apparently healthy condition. It then grows rapidly and nothing more is observed to remind one of the trial through which the animal has passed until it comes to breeding age. Then we find, as shown in the data in Herd B, that the breeding efficiency of the young animal goes back at once to the health of the young calf; if the calf was healthy and its exposure to infection limited, its breeding efficiency will be comparatively high; if ill as a young calf, its breeding efficiency will be low.

Some breeders who are very observant find that heifer calves which are born prematurely at, say, seven to eight months, almost uniformly abort or calve prematurely in first pregnancy. This has so impressed some very observant breeders that they do not permit a calf born prematurely to live, but destroy it at once, not because they can not grow the calf, but because when grown it will be worthless. This observation is thoroughly in harmony with the foregoing, as the prematurely born calf is inevitably highly infected and, although it may apparently recover its health, nevertheless that infection is planted deeply within its system and persists up to and beyond the first breeding year. In fact, we do not know how far beyond the first breeding year it goes. It is a question which requires much further study. For example, in Chart II, in our research animal Number 34, which we infected experimentally by injecting living cultures into her jugular vein when she was carrying in utero our Number 101, the infection was possibly transmitted in utero to Number 101 and is still pursuing her relentlessly, after an interval of four years. The one example does not establish a principle, but a careful study of the chart opens up a very serious question which deserves far more attention than has yet been allotted to it.

The evidences of the importance of maintaining the health of young calves became so great that we decided to undertake researches with a view to determining the precise nature of the infection and how the infection might be avoided; or, when exist-

ing, how it might be overcome or ameliorated. We were soon offered an opportunity to take up this work in a highly valuable breeding herd, which we shall designate Herd D. The herd consists of highly valuable registered Holsteins, in which abortion, sterility and retained afterbirth in cows and white scours and pneumonia in calves have long been a serious menace. Added to this, tuberculosis exists in the herd, although in what degree we have not been advised. Because of the tuberculosis in the herd, the management desired to feed the calves upon pasteurized milk. As in most herds, so in this one, we have no definition to offer for the word "pasteurized." The pasteurization was presumably carried on about as usual, but with just what accuracy and with what effect upon the milk we do not know. By referring to Table A, it will be observed that the mortality from scours and pneumonia was very high, and furthermore that at those times when abortion was common, calf scours and pneumonia tended very strongly to break loose and complete the destruction which the abortion had so well begun. In other words, there is a close relationship between the prevalence of abortion and that of the mortality from white scours and pneumonia. Having been approached by the management for advice, we asked and were granted permission to carry on some definite experiments. Unfortunately, as the herd is located at a distance of some three hundred miles, we could not give that close personal attention to the research work which we desired, and the work was necessarily discontinued before we had carried it far enough to meet the demands thoroughly. However, we obtained numerous interesting data. The management extended every possible courtesy and co-operated heartily.

We desired to use in our researches a large amount of calf scours serum, and preferred to secure this from commercial houses, thus using products which are commonly available to breeders and dairymen in the open market. For this purpose we approached the well-known houses of Parke, Davis & Company and H. K. Mulford Company, who very courteously furnished us with all the serum which we desired. The serum of Parke, Davis & Company is made by immunizing horses, using as an antigen various strains of the colon bacillus and other bacteria obtained from the

feces of calves affected with scours. The Mulford serum differs from that of Parke, Davis & Company in that the cow is used for immunization instead of the horse and the abortion bacillus is added to the colon and other organisms associated therewith. Securing an abundance of the serum, we planned experiments, in outline, as follows:

(1) We procured from the owners a list of the pregnancies terminating January 1, 1915, to June 1, 1916, at which time practically our experiment began. As accurately as possible, we have recorded in Table A the vital statistics of the calves born in this herd during the time named. When the research work began, every calf which remained in the calf stable was ill. The entire lot was unthrifty. Their bowels were irregular, commonly somewhat diarrheic, though the feces as a rule were not very thin, but they varied greatly from day to day. At all times, however, the feces were very sticky and adhered to the tail and thighs. All the calves were coughing more or less. They were hidebound and either pot-bellied or gaunt, and the hair was without lustre. Some of them were markedly ill.

(2) It was decided to select five of the calves which were suffering markedly from scours, including one severe case of pneumonia, and try the effects of calf scours serum upon them. We were here entering a field of research which, so far as we know, had not been previously exploited. The calf scours serum has ordinarily been used as a last resort in highly acute cases of scours and has not been studied in older calves with such chronic lesions as old standing pneumonia, arthritis, and rachitic enlargements. We submit briefly, in the form of tables (Table B) the results upon these calves. It will be observed that in all cases there were distinct improvements, which we could attribute to nothing except the administration of the calf scours serum. The serum appeared to exert a direct and specific influence upon the course of the disease, though necessarily the response could not be so prompt and spectacular in a way as in case of acute disease. Whatever the change in the condition of the animal, it must unavoidably be gradual. Still, the improvement was more marked and far more prompt than we had anticipated.

(3) All cows in the herd which were well advanced in pregnancy, nine, received hypodermically large quantities of the serum (Table C). Following the birth of the calves, we administered to each immediately large doses of the serum, accompanied by high enemas, consisting of a 1 per cent salt solution, to which was added frequently one-fourth per cent of Lugol's solution. Approximately one-half gallon was used as an enema to each calf. The apparatus used consisted of an ordinary hospital irrigator to which was attached a pure gum horse catheter. The horse catheter — soft, pliable, smooth, and well-rounded at the end — was introduced gradually into the rectum while the enema was permitted to flow. By gradually advancing and sometimes slightly withdrawing, and by rotating the catheter upon its long axis, we generally succeeded in introducing it for a distance of eighteen to thirty inches, passing by much of the meconium. The first enema was given as soon as possible after the calf was born, so that the large intestine might be well emptied of the meconium at the beginning. The enema was repeated twice daily. It was found, by this course, that by catching the feces in a vessel while they were being expelled, we could avoid any soiling whatever of the bedding in the stall. The bowel was so thoroughly emptied that the calf did not defecate at other times. The enemas were continued for four or five days. The calves were given at birth two doses — 20 cc. — of serum hypodermically, and thereafter each calf was given one dose — 10 cc.— twice daily, until four days old, so that in the four first days the calf received 90 cc. of the calf scours serum. The serum was then discontinued, to be renewed again should the calf show any scours, arthritis, or other symptoms suggesting the arousal of the infection. The subjoined Table C gives in brief the amount of serum administered to the dams and their calves, with notes recording any marked abnormalities in the behavior of the calf. The results, as here recorded, show some things quite clearly and others dimly. In the first place, it is shown quite clearly that an enormous amount of the serum may be given to pregnant cows and to new-born calves without injury. In fact, there is but one record of apparent injury as a result of the administration of the serum to cows. In that one instance there appeared to be a loss of appetite in the pregnant cows which had

received the M. serum, but this continued for a few hours only, and there was no reappearance of it after succeeding administrations. It is not certain just what caused this apparently slight indisposition. In another case, one of the calves after having received the serum, showed a very marked edematous swelling of the eyelids. We could not determine, however, whether the swelling had been caused by the disease or by the serum. Aside from these two slight irregularities, nothing was observed which could possibly be attributed to the ill effects from the administration of the serum.

TABLE A

RECORD OF THE TERMINATIONS OF PREGNANCIES IN HERD D, JANUARY 1, 1915, TO MAY 21, 1916, INCLUSIVE

Serial number	Age in years	Pregnancy terminated	Result	Health of Calf		Age in days at death	Remarks
				At birth	At 2 days		
1.....	6	1- 3-15	X. R.	Very weak	=	10	
2.....	3	1- 9-15	C	H	H		
3.....	2	1-11-15	C	H	H		
4.....	4	1-25-15	C	H	H		
5.....	9	2- 1-15	C	H	H		
6.....	3	2- 7-15	C	H	H		
7.....	6	2-12-15	C	H	H		
8.....	6	2-12-15	C	H	H		
9.....	2	2-15-15	A			A	Still birth.
10.....	6	2-27-15	C	H	H		
11.....	6	3- 8-15	C	H	H		
12.....	5	3-14-15	C	H	H		
13.....	5	3-24-15	C	H	H		
14.....	6	3-25-15	C	H		150	Rachitis.
15.....	2	3-28-15	C	H	H		
16.....	9	4-16-15	C	H	Prostrate (1)	5	
17.....	5	4-20-15	C	H	Prostrate	No record	Rachitis.
18.....	4	4-28-15	C	H	H	240	Gen. sepsis.
19.....	2	5- 8-15	C	H	H		
20.....	2	5-14-15	C	H	H		
21.....	3	5-25-15	C	H	Stiff		Rachitis.
22.....	5	6- 4-15	C	H	H		
23.....	5	6- 7-15	C	H	H		
24.....	5	6- 8-15	C	Sc		2	
25.....	4	6-15-15	C. R.	Sc	Sc	3	
26.....	2	6-18-15	C	H	H		
27.....	2	6-21-15	C	H	H		
28.....	6	7-29-15	C. R.	—	—	7	
29.....	4	8 -3-15	C	H	H		
30.....	6	8-14-15	C	H	H		
31.....	5	8-20-15	C	H	H		
32.....	8	8-24-15	C	Arthritis	Arthritis	Recovered after 10 days.	
33.....	6	8-25-15	C	Sc	Sc	2	Cow died (sepsis) 2 days after calving.
34.....	6	8-25-15	C	Sc	Sc		

TABLE A — (*Continued*)

Serial number	Age in years	Pregnancy terminated	Result	Health of Calf		Age in days at death	Remarks
				At birth	At 2 days		
35.....	7	9– 3–15	A. R.			A	
36.....	2	9– 5–15	A. R.			A	
37.....	10	9– 9–15	C	Pneumonia	=	11	
38.....	6	9–12–15	C	H	Umb.	8	
39.....	3	9–16–15	C	H	H		A. at 2 yrs. also.
40.....	2	9–20–15	C	H	Acute sepsis	8	
41.....	2	9–20–15	A			A	
42.....	9	10– 5–15	C. R.	H	Sc	4	
43.....	3	10– 8–15	C	H	H		
44.....	3	10–11–15	A. R.			A	
45.....	11	10–13–15	C	H	H		
46.....	11	10–13–13	C	H	Sc	13	
47.....	7	10–14–15	C	Sc	Pn.		
48.....	6	10–15–15	C	H	Umb.	7	
49.....	4	10–18–15	C	Sc	H		
50.....	5	10–20–15	C	H	H		
51.....	9	10–20–15	C	Sc	Sc and Umb.		
52.....	6	10–22–15	C	Sc	Sc and Pn.		
53.....	7	10–27–15	C	Sc	Sc	15	
54.....	5	11– 1–15	C	Sc	Sc		
55.....	6	11–26–15	C	Sc	Sc		
56.....	11	12– 2–15	C	Sc		1	Cow A. 1914.
57.....	6	12– 3–15	A. R.			A	
58.....	4	12– 4–15	A			A	A. at 6 mos.
59.....	2	12–14–15	A			A	A. at 7 mos.
60.....	5	12–16–15	C	Sc	Sc		
61.....	6	12–22–15	C	Sc	Sc	5	
62.....	4	12–30–15	A. R.			A	
63.....	3	12–30–15	C	Sc	Sc		
64.....	2	1– 4–16	A			A	
65.....	6	1– 8–16	C	Sc			
66.....	2	1–12–16	C	Sc	Sc	3	
67.....	5	1–14–16	X. R.	Sc	H		
68.....	5	1–15–16	C	Sc	H		
69.....	4	1–22–16	C	Sc	H		

TABLE A — (*Concluded*)

Serial number	Age in years	Pregnancy terminated	Result	Health of Calf		Age in days at death	Remarks
				At birth	At 2 days		
70.....	4	1-27-16	C	Sc	H		
71.....	8	1-28-16	C	Sc	H		
72.....	6	2- 7-16	C	Sc	H		
73.....	2	2- 7-16	C	Sc	H		
74.....	4	2-11-16	C	Sc		2	
75.....	4	2-29-16	C	Sc	H		
76.....	7	3- 2-16	C	Sc	H		
77.....	6	3-11-16	C	Sc	H		
78.....	6	3-23-16	C	Sc	Sc	3	
79.....	2	3-25-16	C	Sc	Sc	11	
80.....	2	3-29-16	C	Sc	Sc	3	
81.....	7	4- 4-16	A			A	
82.....	7	4- 5-16	C	Sc	Sc		Relapse of Sc.
83.....	4	4- 9-16	A			A	7 mos. fetus. Lived 15 min.
84.....	3	4-10-16	C	Sc	Sc	3	
85.....	6	4-21-16	C	Sc	Sc. Umb.		
86.....	5	4-23-16	A			A	
87.....	2	5- 6-16	C	Sc	Sc	5	
88.....	6	5-10-16	C	Sc	Sc		
89.....	6	5-19-16	C	Sc	Sc	5	
90.....	8	5-19-16	C	Sc	Sc	5	
91.....	8	3- 5-16	C	Sc	Pn		
92.....	4	4-23-16	C	Sc	Pn. & Constip		
93.....	7	5-21-16	C	Sc	Sc		
94.....	6	3-15-16	C	Sc	Sc		
95.....	6	5-12-16	C	Sc	Sc & Pn.		
96.....	6	4——16	C	Sep.	Sep.	7	

A=Abortion.
C=Calved.
X=Premature birth.
R=Retained afterbirth.

H=Healthy.
Sc=Scours.
Umb=Umbilic infection.
Pn=Pneumonia.

TABLE B

Notes Upon Five Calves Recorded in Table A, Born Prior to the Beginning of Experiments with Calf Scours Serum

Number 91 of Table A

Date	General condition	Serum given	Enemas given
Born 3- 5-16	Suffered immediately after birth from scours to which pneumonia was later added.		
6- 2-16	Severely ill from pneumonia. Scours still present. The calf was weak, emaciated, dull, breathing rapidly, and its tail and buttocks were smeared with feces. Temperatures 103.1, 103.6 and 104.4.	3 P. M.—20 c.c.P. 8:45 P. M.—10 c.c.P.	
6- 3-16	Calf appears bright. Temperatures 102.6, 102 and 101.6.	8:30 A. M.—10 c.c. 8:30 P. M.—10 c.c.	
6- 4-16	Temperature, 101.3.	9:00 A. M.—10 c.c.	
6- 5-16	Temperature, 100.5.	9:30 A. M.—10 c.c.	
6- 6-16	Appears very well. Temperature, 99.5.	9:00 A. M.—10 c.c.	
6- 7-16	Normal temperature. Treatment discontinued.		
6- 8-16	Normal with good appetite. A skin eruption on nose, shoulders and neck.		
6-16-16	Normal in every respect. The eruption treated antiseptically and is well under control.	Total serum given, 80 c.c.	

TABLE B — (*Continued*)

Number 92 of Table A

Date	General condition	Serum given	Enemas given
6– 3 16	Bowels torpid. Coat very rough. Respiration quickened and jerky. Pneumonia.	10 c.c.P.—8 A. M. 10 c.c.P.—8 P. M.	
6–14–16	Calf decidedly improved.	10 c.c.P.—8:30 A. M. 10 c.c.P.—6 P. M.	
6–15–16	Temperature, 103.1. Feces somewhat hard.	10 c.c.P.—A. M. 10 c.c.P.—P. M.	
6– 6–16		10 c.c.P.	¼% Lugol's in 1% salt.
6– 7–16	Bowels greatly improved. Respirations slightly jerky. Calf feeling fine and taking food with avidity.	Total, 70 c.c.	
6–16–16	Calf well.		

TABLE B — (*Continued*)

Number 93 of Table A

Date	General condition	Serum given	Enemas given
5–29–16	Feces soft and mixed with some blood. Calf had previously had severe scours, and was at times comatose. At 5:30 P. M. calf very dull, comatose, and could not get up. Appetite good. At 9 P. M. acute tarsitis, joint very tender, calf would bear no weight on limb. Calf bright and hungry.	20 c.c.P.—5:30 P. M.	1% salt and 0.25% Lugol's at 5:30 P. M.
5 30–16	Calf continued very lame in morning. Lower eyelids swollen and edematous. At 9 A. M. feeling very much better, tarsitis receding so calf walked comfortably. Took 1 pint milk with avidity every 3 to 4 hours.	10 c.c.P.—A. M.	
5–31–16	Feces quite soft and contained an increased amount of blood. Calf appeared much better. Lameness almost entirely gone. Swelling receding.		
6– 1–16	Condition highly satisfactory.		
6– 2–16	At 3 P. M. tail soiled with dry feces. Calf passed small amounts of blood.	10 c.c.P.—3 P. M.	
6– 3–16	At 8:30 A. M. feces moderate in volume, firm, greenish yellow, and not bad odor, contained some blood. Calf bright. At 8:30 P. M. calf playing vigorously.	10 c.c.P.—8:30 A. M.	
6– 4–16	Calf bright.		
6– 5–16		10 c.c.P.—8 P. M.	
6– 6–16		10 c.c.P.	1% salt and 0.25% Lugol's.
6– 7–16	2 movements of bowels of 1 oz. each, of olive color and containing some blood.	20 c.c.P.—A. M. 20 c.c.P.—P. M.	
6–16–16	Improving slowly.	10 c.c.P. and enema on alternate days.	

TABLE B — (*Continued*)

Number 94 of Table A

Date	General condition	Serum given	Enemas given
5–29–16	Has had dysentery intermittently since birth. Feces bloody at times.	10 c.c.P.	
5–31–16	Dysentery absent since 29th. Feces normal.		
6– 4–16	6 P. M., condition of calf remained indifferent and unthrifty.	10 c.c.P.	
6– 5–16	9:30 A. M., feces brownish in color, normal in consistence and odor.	10 c.c.P.	
6– 6–16	8 P. M., doing well.	10 c.c.P.	
6– 7–16	Bowels normal. The diet, which had been restricted, was ordered gradually increased. Erosions in nasal mucosa just inside nostrils. Small acne in skin about nostrils.	10 c.c.P.	
6– 8–16 to 6–13–16	Dysentery returned. Two 10 c.c. doses of serum given daily until June 13, when it was discontinued. At that time the dysentery was still present.		
6–16–16	Bowels very loose. Not improving.		

TABLE B — (*Concluded*)

Number 95 of Table A

Date	General condition	Serum given	Enemas given
Born 5-12-16	Had scours constantly since birth.		
5-29-16	Feces of grayish color and semi-solid.		High enema with 0.25% Lugol's.
5-31-16	Feces brownish color and better consistency. Improved.		
6- 2-16	Calf feeling well, though scouring some, soiling tail and buttocks.	10 c.c.P.—3 P. M.	
6- 3-16	Looking and feeling well. No scours observed.	10 c.c.P.—8:30 A. M. 10 c.c.P.—8:30 P. M.	
6- 4-16	Calf looking well, but feces were fetid.	10 c.c.P.—A. M. 10 c.c.P.—P. M.	Enema.
6- 5-16	General condition fair. Feces soft yellowish brown. Temperature 103. Breathing accelerated.	10 c.c.P.—9:30 A. M. 10 c.c.P.—8 P. M.	
6- 6-16	Three ounces feces passed, light brown in color, fetid. Calf very bright, looking well. Temperature 102.	10 c.c.—A. M. 10 c.c.—P M.	
6- 7-16	Respiration still slightly accelerated. Temperature 100.2. Feces thin, brownish grey. Calf looked and ate well.	10 c.c.P.—A. M. 10 c.c.P.—P. M.	
6-16-16	Calf doing well.	Total, 110 c.c.	

TABLE C

NOTES UPON NINE COWS GIVEN CALF SCOURS SERUM PRIOR TO CALVING, WITH THEIR CALVES, AND ONE ADDITIONAL CALF

Number 97

Date	Serum given	Calved	Expelled afterbirth	Remarks
5-29-16	10 A. M.—30 c.c.P. 10 P. M.—20 c.c.P.			
5-30-16	11 A. M.—20 c.c.P. 9 P. M.—10 c.c.P.			
5-31-16	9 A. M.—20 c.c.P.			
6- 2-16	Total, 100 c.c.	Unseen, probably 2 or 3 A. M. Calf dry and had sucked when first seen.	Before 7 A. M., probably at 4 hours.	

Number 97's calf

Date	General condition	Serum given	Enemas given
6- 2-16	Feces discharged after enema copious, bright green. At 10 A. M. further copious discharge of intensely green feces, soft, free from gas bubbles, odorless. 11 A. M., small passage of bright yellow feces.	6 A. M.—20 c.c.P. 8:30 P. M.—10 c.c.P.	Normal salt solution + .25% Lugol's solution 9 A. M.
6- 3-16	Enema brought away a copious discharge of bright yellow feces of soft consistence, good odor.	8:30 A. M.—10 c.c.P. 8:30 P. M.—10 c.c.P.	Enema 4 P. M.
6- 4-16	Feces normal yellow.	8:30 A. M.—10 c.c.P.	Enema 8:30 A. M.
6- 5-16	No natural movement as yet.	Morning —10 c.c.P.	
6- 6-16	No natural movement. Feces rich yellow color from enema.		
6- 7-16	Calf feeling fine. No natural bowel movement as yet.		

TABLE C — (*Continued*)

Number 98

Date	Serum given	Calved	Expelled afterbirth	Remarks
5–29–16	10 A. M.—30 c.c.P. 10 P. M.—20 c.c.P.			
5–30–16	11 A. M.—20 c.c.P. 9 P. M.—10 c.c.P.			
5–31–16	A. M.—20 c.c.P.			
6– 1–16		At 12:30 P. M.	Cleaned promptly.	
6– 5–16	A. M.—20 c.c.M. P. M.—20 c.c.M.			Sanguinous vaginal discharge.
6– 6–16	A. M.—20 c.c.M. P. M.—20 c.c.M.			
6– 7–16	A. M.—20 c.c.M.			
	Total, 200 c.c.			

TABLE C — (*Continued*)

Number 98's calf

Date	General condition	Serum given	Enemas given
6– 1–16	Apparently well.	1 A. M.—20 c.c.P.	
6– 2–16	Feces evacuated by high enema were of normal volume, soft, yellow, and of normal odor.	9 A. M.—10 c.c.P. 8:30 P. M.—10 c.c.P.	High enema.
6– 3–16	Calf bright. No feces since previous enema, which brought away large volume of yellow feces of good odor.	8 A. M.—10 c.c.P. 8:30 A. M.—10 c.c.P.	Enema at 4 P. M.
6– 4–16	Enema brought forth brownish-yellow feces.	8 A. M.—10 c.c.P. 8 P. M.—10 c.c.P.	
6– 5–16	Six ounces brownish feces of good consistency, tinged slightly at one point with blood, were voided.	9 A. M.—10 c.c.P. 8 P. M.—10 c.c.P.	
6– 6–16	Calf feeling fine. One ounce feces, brown in color, normal in appearance, voided naturally.		Enema at noon.
6– 7–16	No feces observed. Calf well.		
6–16–16	Calf doing finely.		
7– 7–16	Calf not doing well. Dull. Ears drooped. Temperature 103.4. Feces very soft, sticky, light in color, and very offensive.		Enema daily.
7–14–16	Calf somewhat better. Temperature 102.3 to 103.3. Appetite good, bowels in good condition. Used camphorated oil and brandy stimulant.		
7–28–16	Greatly improved. Bowels and temperature normal.		

TABLE C — (*Continued*)

Number 99

Date	Serum given	Calved	Expelled afterbirth	Remarks
5–29–16	6 P. M.—20 c.c.M.			
5–31–16	9 A. M.—20 c.c.M.			
6– 1–16	10 c.c.M.			
6– 2–16	20 c.c.P.			
6– 3–16	20 c.c.P.			
6– 4–16	20 c.c.P.			
6– 5–16	20 c.c.P.			
6– 6–16	20 c.c.M.			
6– 7–16	20 c.c.M.			
6– 8–16	20 c.c.M.			
6– 9–16	20 c.c.M.			
6–10–16	20 c.c.M.			
6–11–16		After one hour was assisted in expelling calf.	In eight hours.	

TABLE C — (*Continued*)

Number 99's calf

Date	General condition	Serum given	Enemas given
6–11–16	Calf well.	A. M.—20 c.c.M. P. M.—10 c.c.M.	High enemas morning and evening for four days.
6–12–16	Calf well.	A. M —10 c.c.M. P. M.—10 c.c.M.	
6–13–16	Calf well.	A. M.—10 c.c.M. P. M.—10 c.c.M.	
6–14–16	Calf well..	A. M.—10 c.c.M. P. M.—10 c.c.M.	
6–15–16	Calf well.		
6–16–16	No symptoms of scours so far.		

TABLE C — (*Continued*)

Number 100

Date	Serum given	Calved	Expelled afterbirth	Remarks
5–29–16	A. M.—10 c.c.P. P. M.—10 c.c.P.			
5–30–16	10 c.c.P.			
5–31–16	20 c. c.P.			
6– 1–16	20 c.c.P.			
6– 2–16	20 c.c.P.			
6– 3–16	20 c.c.P.			
6– 4–16	20 c.c.P.			
6–16–16	20 c.c.P.			
6–17–16	20 c.c.P.			
6–18–16	20 c.c.P.			
6–19–16	20 c.c.P.			
6–20–16	20 c.c.P.			
6–21–16	20 c.c.P.			
6–22–16	20 c.c.P.			
6–23–16	20 c.c.P.			
6–24–16	20 c.c.P.			
6–25–16	20 c.c.P.			
6–26–16		Calved.		

TABLE C — (*Continued*)

Number 100's calf

Date	General condition	Serum given	Enemas given
Born 6–26–16	Good. Well and vigorous until 6–29–16.	A. M —20 c.c.M. P. M.—10 c.c.M.	High enemas morning and evening for 4 days.
6–27–16		A. M —10 c.c.M. P. M.—10 c.c.M.	
6–28–16		A. M —10 c.c.M. P. M.—10 c.c.M.	
6–29–16	Extreme weakness. Could barely get up alone and was unsteady in gait. Temperature 104.4.	A. M —10 c.c.M. P. M.—10 c.c.M.	
7– 3–16	Greatly improved. Very little lameness or weakness, though stiff. Temperature 103.3. Calf did not appear sick. Bowels in good shape.		
7– 7–16	Calf doing all right. Temperature normal.		
7–14–16	Temperature 104.3. Feces soft, but not scouring.		
7–28–16	Condition greatly improved. Bowels and temperature normal.		

TABLE C — (*Continued*)

Number 101

Date	Serum given	Calved	Expelled afterbirth	Remarks
5–29–16	20 c.c.M.—6 P. M.			
5–31–16	20 c.c.M.—9 A. M			
6– 1–16	10 c.c.P.			
6– 2–16	20 c.c.P.			
6– 3–16	20 c.c.P.			
6– 4–16	20 c.c.P.			
7– 8–16		Calved.		

Number 101's calf

Date	General condition	Serum given	Enemas given
7– 8–16	Calf well.	20 c. c.M. at birth and one 10 c.c. dose morning and evening for 4 days.	High enema morning and night for 4 days.
7–14–16	Calf doing well.		
7–28–16	Calf doing finely.		

TABLE C — (*Continued*)

Number 102

Date	Serum given	Calved	Expelled afterbirth	Remarks
5–29–16	20 c.c.M.—6 P. M.			
5–31–16	20 c.c.M.—9 A. M			
6– 1–16	10 c.c.M.			
6– 2–16	20 c.c.P.			
6– 3–16	20 c.c.P.			
6– 4–16	20 c.c.P.			
7– 8–16		Calved.		

Number 102's calf

Date	General condition	Serum given	Enemas given
7– 8–16	Calf well.	20 c.c.P. serum at birth and 10 c.c. doses morning and night for 4 days.	?
7–14–16	Calf doing well.		?
7–28–16	Calf doing finely.		

TABLE C — (*Continued*)

Number 103

Date	Serum given	Calved	Expelled afterbirth	Remarks
7–10–16	A. M.—20 c.c.M. P. M.—20 c.c.M.			
7–11–16	A. M —20 c.c.M. P. M.—20 c.c.M.			
7–12–16	A. M —20 c.c.M. P. M.—20 c.c.M.			
7–13–16	A. M.—10 c.c.M. P. M.—10 c.c.M.			
7–14–16	A. M —10 c.c.M. P. M.—10 c.c.M.			
7–15–16	A. M.—10 c.c.M. P. M.—10 c.c.M.			
7–16–16	A. M.—10 c.c.M. P. M.—10 c.c.M.			
7–19–16	Total serum, 200 c.c.	Slight assistance given after about three-quarters of an hour.	7 hours after calving.	

TABLE C — (*Continued*)

Number 103's calf

Date	General condition	Serum given	Enemas given
7–17–16		20 c.c.M.	Enema.
7–20–16		10 c.c.M.	Enema.
7–21–16		10 c.c.M.	Enema.
7–22–16		10 c.c.M.	Enema.
7–28–16	Has never shown any symptoms of scours.	Total, 50 c.c.	

TABLE C — (*Continued*)

Number 104

Date	Serum given	Calved	Expelled afterbirth	Remarks
7–21–16 to 7–25–16 incl.	150 c.c.M.			
7–29–16		Unusually large calf. Assisted after 1¼ hours.	In 9 hours.	

Number 104's calf

Date	General condition	Serum given	Enemas given
7–29–16		A. M.—10 c.c.M. P. M.—10 c.c.M.	Enema.
7–30–16		10 c.c.M.	Enema.
7–31–16		10 c.c.M.	Enema.
8– 1–16		10 c.c.M.	Enema.
8–12–16	Calf doing finely.		

TABLE C — (*Continued*)

Number 105

Date	Serum given	Calved	Expelled afterbirth	Remarks
7–21–16 to 7–25–16 incl.	160 c.c.M.			
8– 1–16		In 1¼ hours. No assistance.	3 hours.	

Number 105's calf

Date	General condition	Serum given	Enemas given
8– 1–16		A. M —10 c.c.M. P. M.—10 c.c.M.	Enema.
8– 2–16		10 c.c.M.	Enema.
8– 3–16		10 c.c.M.	Enema.
8–12–16	Calf doing well.	Total, 40 c.c.	

TABLE C — (*Concluded*)

Calf number 106

Date	General condition	Serum given	Enemas given
Born 7–23–16			Enema.
7–24–16		A. M.—20 c.c.M. P. M.—10 c.c.M.	Enema.
7–25–16		A. M.—10 c.c.M. P. M.—10 c.c.M.	Enema.
7–26–16		A. M.—10 c.c.M. P. M.—10 c.c.M.	Enema.
7–28–16	Calf doing well.		

Studying Table A, and the succeeding Tables B and C, of the calves and cows placed under treatment, it will be observed that there was a sharp check in the mortality among the calves as soon as the serum was administered. In fact, after the experiment began on June 1, until late in August, when the researches were discontinued, there was not a fatality among the calves in the herd. This is in very sharp contrast to the previous conditions. The serum was given a very trying test. The calves remained in the same stable where the 98 calves recorded in the chart had been kept from birth. The new-born calves listed in our experiment were placed in this thoroughly infected stable among the older calves which were still highly infected and ill from the disease. The stable was kept clean, of course, and some effort was made to disinfect. The stable is admirably constructed and ventilated, and no expense has been spared in making it as good as the designer considered possible. It has, however, the lamentable defect that the stalls are constructed of woven wire, so that essentially all the calves are in one room and infection has free play in passing from one stall to another. The stable could not be thoroughly disinfected because the sick calves remained in it and continued as a center of infection, however much work might be done in the stable. The feeding of the calves was also continued on practically the same lines. The pasteurization of the milk went on the same as before, and no material change in any essential respect was made, except the three items of administering the serum to the pregnant cows and to the sick or to the new-born calves and the administration of the enemas to the calves.

It is reasonably clear — at least, to our minds — that the administration of the serum and of the enemas, combined or alone, brought about an abrupt change in the calves. Time and material were wanting to determine just what part each of the three elements played in checking the mortality. In many respects, however, the results came out with reasonable clearness. In the calves already sick at the time that the experiment began, the effect of the serum was clear and distinct. It was clearer to one who could watch the calves in the stable than it can be to one who can only read the results.

The effect of the use of the serum upon the pregnant cows is less clear. Prior to the administration of the serum to this group

of cows, most of the cows in which pregnancy was terminating suffered quite severely from metritis, accompanied by retained afterbirth. After the administration of the serum, the metritis appeared to abate definitely. The uterus contracted more vigorously, so that the calf was born more promptly and the afterbirth was expelled in less time than prevailed as a rule prior to the beginning of the experiment. It is not easy, however, to attribute this definitely to the use of the serum, because metritis goes in waves in any herd, and it is perfectly possible that it may have been time for the prevalence of the disease to abate. It would therefore be quite unsafe to draw any definite conclusion with reference to the value of this product administered to pregnant cows. It should be remembered, however, as we have stated before, that the infection which exists in the uterus in cases of metritis and that which exists in the meconium of the new-born calf, causing scours are, so far as we can determine at present, identical in character. It is therefore only logical to suspect that if the serum can exert a specific effect upon this infection when it is causing white scours it should also in a measure affect favorably the same infection when it exists in the uterine cavity of the pregnant cow. This appears to hold true.

When a calf is born of a cow which has received this serum during the latter part of her pregnancy and the calf is then given large doses of the serum, it is impossible to determine from a single experiment which of the two processes had the greater influence on the calf. Neither do we know what part the enemas played in the protection of the calf. We are strongly inclined to believe that each of the three elements played an important part, though just what relationship they bear to one another remains to be determined.

Some light may be thrown upon this question by our research calves Numbers 15, 16 and 17, notes of which are appended (Table D). Number 15 was dropped by a tuberculin reactor which was awaiting slaughter. The calf remained with the dam for about an hour, and presumably sucked. She was then fed upon raw milk, at first from a cow which was considered healthy, Number 49 of Chart II, and later upon the milk from our Number 34, which we knew to be highly infected with contagious

TABLE D

NOTES UPON THE USE OF CALF SCOURS SERUM UPON THREE CALVES IN RESEARCH HERD

Number 15, calf from a tuberculin reactor, fed upon raw milk from non-tubercular cows.

Number 16, dam seriously ill after calving from metritis with retained fetal membranes and gangrene of all cotyledons. Calf fed upon boiled milk. Meconium of calf highly infected.

Number 17, dam died from general sepsis due to metritis existing before calving. Blood of calf reacted strongly to agglutination test (1–100).

Number 15

Date	General condition	Serum given	Enemas given
Born 10–10–16	Good, healthy, strong calf.		
11– 2–16	Calf has cough, mucoid nasal discharge, and fetid diarrhea. Coat rough and staring, but in spite of all this appetite good.	A. M.—20 c.c.P. P. M.—10 c.c.P.	High enema, salt solution.
11– 3–16	Appearance still poor. Temperature 106.3. No diarrhea. Feces fetid, but fairly firm.	A. M.—20 c.c.P.	High enema.
11– 4 16	Much improved. Feces removed were voluminous and very ill smelling. Temperature 105.7.		High enema.
11– 5–16	Condition better. Feces still fetid. Temperature 105.		Enema containing ½% Lugol's.
11–06–16	Much brighter. Temperature 104.	A. M.—10 c.c.P.	Enema containing ½% Lugol's.
11– 7–16	Much brighter. Temperature 103.7. Feces not so fetid as before.		Enema containing ½% Lugol's.
11– 8–16	Calf feeling good. Feces still fetid. Temperature 103.4.		Enema containing ½% Lugol's.
11–11–16	Calf seemed in distress, breathed hard. Gave enema. Great improvement seemed to follow immediately. Feces fetid, light yellow.	A. M.—10 c.c.P.	
11–12–16	Calf appeared to be feeling well. No treatment.	Total serum, 70 c.c.	

TABLE D — (*Continued*)

Number 16

Date	General condition	Serum given	Enemas given
Born 10–16–16	Apparently normal calf, though born about one week prematurely.		
10–18–16	Did not seem to be doing well. Was depressed and lifeless. Temperature 102.7. Toxaemia?	5 P. M.—10 c.c.P.	High enema, 1% salt solution.
10–19–16	Condition not improved. Character of feces about normal, quite firm.	8 A. M.—20 c.c.P. 5 P. M.—10 c.c.P.	Enema, 0.25 % Lugol's solution..
10–20–16	Condition much better.	A. M.—10 c.c.P. P. M.—10 c.c.P.	Enema, 0.25% Lugol's solution.
10–21–16	Bright and lively.	A. M.—10 c.c.	Enema.
10–24–16	Gaining flesh rapidly. Feces normal.		Enema.
10–25–16	Treatment discontinued.	Total, 70 c.c.	

TABLE D — (*Concluded*)

Number 17

Date	General condition	Serum given	Enemas given
Born 11– 3–16	Calf seemed normal, but was never very lively.		
11– 6–16	Calf seemed duller than usual. Enema brought forth some fetid feces, slightly blood-stained. Not observed to be lame at 8 A. M., but at 11 A. M. was intensely lame in left hock.	11:30 A. M.—20 c.c.P. 5:30 P. M.—20 c.c.P.	High enema of 1% salt solution.
11– 7–16	Dull. Temperature 104.7. Lameness not so marked. General condition unimproved. Feces contained blood specks, were very fetid, but quite firm.	8:30 A. M.—10 c.c.P. 5:30 P. M.—10 c.c.P.	High enema.
11– 8–16	Still sluggish. Feces a little softer, but do not have an especially offensive odor. Temperature 104.8. Lameness gone.	A. M.—10 c.c.P. P. M.—10 c.c.P.	High enema.
11– 9–16	Marked depression. Temperature 105.	A. M —20 c.c.P. P. M.—10 c.c.P.	High eenma.
11–10–16	Condition much improved. Temperature 102.8 and 103.	A. M.—10 c.c.P. P. M.—10 c.c.P.	High enema.
11–11–16	Improving. Temperatures 103 and 104.6.	A. M.—10 c.c.P. P. M.—10 c.c.P.	High enema.
11–12–16	Appears to be feeling well.		High enema.
11–16–16	Lame in left hock. No swelling, but great tenderness on pressure. No weight borne. Feces very fetid.	P. M.—20 c.c.P.	High enema containing .05% Lugol's.
11–17–16	Lameness still present. Feces fetid, fairly firm, and specked with blood. Temperature 104.4.	P. M.—20 c.c.P.	High enema containing .05% Lugol's.
11–18–16	Lame. Othermise well. Free passage normal feces.	A. M.—10 c.c.P.	
11–19–16	Lameness disappearing. Feeling well.	A. M.—10 c.c.P.	
11–21–16	Only a trace of lameness.	A. M —10 c.c.P.	
11–21–16	Calf normal, feeling fine.	Total, 220 c.c.	

abortion. It will be observed from the chart that she suffered repeatedly from scours and that as often as she suffered the scours was controlled very promptly by the use of the serum. The high enemas were used in this instance, not with a view to any curative effect, but for the purpose of studying the volume and character of the feces, regardless of the effect it might have upon the general well-being of the calf.

Number 16 is the calf of Number 101. She was apparently well at the time of birth, but very quickly became ill and went down in a stupor. She was fed from the first upon boiled milk, except that she was born unseen and may have sucked her dam once. It will be seen that she was quite ill for a time and that she received a large amount of calf scours serum. After five days she recovered completely. Since that time she has grown with remarkable rapidity, averaging over one pound a day upon a comparatively limited amount of milk. She is very vigorous and playful. Her coat is highly lustrous. Her feces are normal and do not adhere in the least to the tail and buttocks. In other words, she keeps perfectly clean without the aid of brushing or sponging.

Research calf Number 17 was born ill. The dam died from general peritonitis as a result of the infection which existed in her uterus before giving birth to the calf. The metritis existing in the uterus was so great that the organ was unable to expel the calf, which was normal in size, in presentation, and in position, but had to be removed by gentle traction simply because of the inertia of the uterus due to the infection present. Calf scours serum was administered promptly, but it required an enormous amount to bring about any amelioration in the symptoms. Like Number 16 she was fed exclusively upon boiled milk. Finally she responded to the treatment, recovered completely, and is now growing rapidly and without interruption, the same as Numbers 15 and 16.

None of the three ever show any soiling of the tail or buttocks from the feces. Each is playful and vigorous. Their coats are highly lustrous. They are neither gaunt nor pot-bellied and are growing vigorously.

These data, though limited in volume, have convinced us that calf scours and pneumonia may be prevented or controlled in essentially all instances. The various manufacturers of calf scours sera presumably use various methods. The two sera which we have used do not readily submit to accurate comparison. In the researches which we have here recorded and in other clinical researches which do not so readily lend themselves to record, we have found that each of these sera, when given liberally, appears to act specifically in halting the course of the disease and bringing about recovery. The specific action of the product seems to be equally marked in both the chronic and the acute infection, though in the latter the results are always more vivid.

We believe also that the high enemas are very beneficial in the preventive and curative handling of scours. We believe that, before a calf is fed milk, the bowels should be thoroughly emptied of the meconium. For this purpose, three or four high enemas may be administered in rapid succession, until it is quite certain that all, or nearly all, of the meconium has been removed and with it has come away much of the infection which exists in the alimentary canal of the calf.

Just what and how much the calf shall be fed is a problem of great importance which it is difficult at present to decide. There can be no question, according to previous researches, which we recorded in our report for 1914–15, that boiled milk is not as safe for a new-born calf as is raw milk. There has been much discussion regarding the comparative safety of pasteurized and boiled milk, and many believe that the former is much safer than the latter. We have been unable to recognize this. Calves have scours, whether fed upon raw, pasteurized or boiled milk. The scours is not so liable to appear when the calves are fed upon raw milk as upon pasteurized or boiled milk, but according to our observations there is absolutely no advantage in feeding pasteurized milk instead of boiled milk. In fact, according to our researches, the boiled milk is the safer of the two, because in pasteurizing milk the process is often incomplete and such restrictive bacteria as the lactic acid bacilli are apparently destroyed while the bacteria of decomposition, such as the colon bacilli, remain alive and play an important part in the engendering of scours.

The explanation of the differences in the effects of feeding raw and boiled milk to calves has not yet been made clear. Some investigators say that it is due to the destruction by boiling of certain enzymes or other organic substances which are essential to digestion; others that the boiling or pasteurizing precipitates and renders insoluble the lime salts within the milk. As a matter of fact, neither of these explanations really meets the situation. In our research herd, when a cow expels a calf in fifteen to twenty minutes and the fetal membranes follow in an hour and a quarter to an hour and a half, and the cow has no further discharge from the genital tract, the calf will live and thrive perfectly from the very outset upon boiled milk. If, however, the cow has metritis during her pregnancy, like the dams of our Numbers 16 and 17, and we attempt to grow the calf upon boiled milk we are certain to have difficulty with calf scours. We are very liable to have that difficulty anyhow, even if we use raw milk, but unquestionably the raw milk is the safer of the two. From our standpoint it would appear that the raw milk contains some of those hypothetical antibodies, something similar to the agglutinins, which are destroyed by heat. If the milk is given raw, these substances restrain or destroy the bacteria present in the alimentary tract. This is made clearer by the researches of Giltner and others showing that the milk of cows infected with contagious abortion contains the agglutinins for contagious abortion — that is, the milk of such cows will agglutinate a suspension of the abortion bacilli.

Reasoning from analogy, it would be quite natural to assume that the colon bacillus, which plays so important a part in the infection of calf scours, has also produced in the milk an effect similar to that of the abortion organism, so that the raw milk contains agglutinins for both the abortion and the colon organisms and when fed to a calf tends to overcome and destroy the bacilli which may be present in the digestive tube of the calf.

The objection to giving raw milk to the new-born calf is that, in spite of any such protective substances which it may possess, it contains also the bacilli themselves or is highly contaminated with bacilli which have been discharged from the genital tract, have flowed down the tail and thighs to the udder, and eventually have been swallowed by the calf in sucking the cow.

In our earlier researches, we lost a number of calves fed upon boiled milk. By our later researches, we believe that those calves might have been saved had we used calf scours serum with sufficient vigor. In such cases, however, as shown in our Number 17, an enormous amount of serum may be required, and when we must pay one dollar for each 10 cc. the cost may quickly become prohibitive upon calves of ordinary value. This will readily be seen from a study of Number 17, for which we expended twenty-two dollars for serum. In our researches we have ignored the question of economics in the administration of calf scours serum, being first of all interested in its power to specifically affect the course of the disease. If found efficient, the economics of its use may be studied out. In order to avoid the practically prohibitive expense of the use of extraordinary quantities of serum we have advised in our recent publications that a calf should be fed upon raw milk for the first eight to ten days of its life. For this purpose we have advised that a healthy cow be selected and that the milk be drawn under the strict precautions laid down for the production of certified milk — that the udder and adjacent parts shall be washed and disinfected before drawing the milk to feed the calf or permitting the calf to suck the cow. After feeding the calf upon raw milk for eight to ten days, whatever virtue there is in raw milk has been fully realized and the calf will grow and thrive upon boiled milk.

The defect in this plan is that the calf inevitably gets with the raw milk considerable infection during the first few days. This is especially true when the herd is tuberculous. If the calves are of sufficient value to warrant the outlay, we feel quite confident that, by using a sufficient volume of serum, from a reliable source, a calf may be fed upon boiled milk from the beginning, but this can apply only to herds of great value. However we may raise a calf, we cannot keep it absolutely free from the infections which cause abortion, white scours and pneumonia, but we can, by reasonable care, avoid the highly injurious infections which generally occur in new-born calves in large dairy and breeding establishments. We feel that in the future the cornerstone of successful breeding and dairying must be the care of the new-born calf. If we desire to grow a healthy and efficient cow, we must, to start

with, have a healthy and vigorous calf, and we must guard this health and vigor throughout the animal's life.

Outline of Recommendations for Control

Readjusted in harmony with the researches for the past few years, my views regarding the source of the infection and its avenue of invasion may be outlined as follows:

1. The largest known volume of the infection accumulates in the gravid uterus and is very largely expelled prior to, during, and soon after the termination of pregnancy.

2. Less in volume than in the uterus, but more frequently recognizable by present methods, is the infection in the milk.

3. The infection may and does pass through the chorion from the utero-chorionic space, penetrates the amniotic cavity, and is swallowed by the fetus. It may cause fetal diarrhœa or may be lodged in the meconium ready to cause white scours or later pneumonia in the new-born calf. Most new-born calves are free from the infection.

4. Infection-free new-born calves generally or always ingest the infection with their milk, either from the interior of the udder or from the exterior of the teat, which has been soiled from the discharge from the genital tract. The latter source is far more dangerous. If the dam or nurse cow is not severely infected, the calf thrives well and presents no evidences of disease: its blood does not react, or reacts very low. If the cow is ill from metritis or retained afterbirth, the intense infection of the calf after birth, as well as before, is more probable, and the severity of the infection tends to be greatly increased. The infection is intensified, especially in dairies, by the use as calf food of unmarketable milk from badly diseased cows. The intensity of the infection is greatly heightened and assured through the feeding of mixed, or composite milk, by which the calf is exposed to the most virulent infection in the herd, and still more seriously when it is fed upon raw skimmed milk and whey from creameries and cheese factories, by which means each calf is exposed to the most virulent strains of the abortion bacillus in the community. It is this exposure which

is chiefly responsible for the constant increase in virulence of the disease in dairies, in contrast with the lesser frequency in beef cattle, where the calf is usually exposed to the milk infection of its dam only.

5. The cohabitation of evidently diseased with apparently sound cows; intermediary bearers, such as attendants and visitors; and the contamination of the food of adult cattle play a minor role in the dissemination of the disease.

6. The bull plays an important role. Definite experimental proof of this is wanting and the clinical evidence is contradictory. The bull must at least be a more probable carrier than an attendant. Logically we can not expect an infection of the genitalia to be unisexual. The bull would naturally tend to be less seriously involved than the cow, and his blood generally reacts more feebly than that of the cow.

7. An abortion storm may be aroused in a herd intrinsically through unfavorable conditions within the herd or extrinsically through the introduction of new cattle of either sex from herds having a more highly virulent type of infection.

These views regarding the source and course of the abortion infection inevitably clash with the old, and still generally held idea of the efficiency of the isolation of aborters in the control of the disease. In my clinical experience handling sterile cows, the frequency with which I find a dead embryonic sac lying in the vagina or in the cervical canal and other evidences of a most convincing kind teach me that probably less than 50 per cent of the actual abortions are seen. Just how anyone can bring himself to believe that isolating 50 per cent of the aborters from a herd will eliminate the disease, I can not understand.

Again, I find a necrotic mass of fetal membranes protruding into the vagina through the cervical canal, and all about it a voluminous discharge taking place. The anterior portions of the fetal sac and the fetus are alive. The discharge from the cervix has evidently existed for weeks. There are no exterior signs of abortion and no rule for isolation. The animal is not known to be an aborter. Finally she aborts a tiny fetus in its membranes and the uterus is at once well nigh clean. Now comes isolation —

if the abortion is discovered, which occurs in less than 1 per cent of such cases. The cow is cleaner and safer than she had been for weeks. I do not understand how isolation, after most of the infection has been discharged, can control the disease.

Sometimes I find a cow carrying a fetal cadaver for one or two years, fetal debris and uterine discharges all the while escaping without attracting attention. She has not aborted; she is in no danger of aborting. She would be more fortunate if she could abort. The isolation scheme does not demand her segregation.

I see cows which carry their calves to full term and expel them alive. For some time prior to calving, they expel pints, quarts, literally gallons of the typical exudate of contagious abortion, but they do not abort. Still, they expel more abortion exudate than twenty cows which abort in early pregnancy. These cows have not aborted and are not subject to quarantine.

Sometimes I see a cow calving prematurely or at full time. Because of the metritis of contagious abortion, she has retained placenta and between the membranes and uterus a great mass of the typical exudate of contagious abortion. Her failure to abort leaves her in the herd, discharging far more virus than most aborters. Her live calf is infected at birth and soon has the dysentery or pneumonia of contagious abortion. Quite naturally the calf has not aborted or been aborted, and under the rules is not subject to quarantine, but it spreads disease and disaster in the calf stable. From such cases down to the point where acceptable evidence of infection vanishes, there is every gradation. The sanitarian who would control contagious abortion by isolation places himself at once between the Scylla of attempting control by removing a pitiable minority of dangerous animals and the Charybdis of practically or completely emptying the stable. The futile process of isolating aborters to control abortion has been the cornerstone in the handling of this scourge for at least fifty years, and the results are so evident to-day that one may well wonder why the plan is still advocated by anyone.

Instead of isolation of aborters, I have advocated for some years a plan of control based upon the conception of the disease outlined above.

(1) Guard and protect the new-born calf. Bathe and disinfect the cow before calving and place her in a clean stall. Remove the calf immediately after birth. Cleanse and disinfect the udder and neighboring parts before permitting the calf to suck or drawing milk for it. Keep the calf upon raw whole milk of the dam or of a selected cow for eight or ten days, and thereafter feed upon sterilized milk, which may be skimmed, mixed, etc. This limits the milk exposure to that from one cow and to the first eight or ten days of the life of the calf.

Keep the calf isolated as long as practicable. If it develops scours or pneumonia, proceed vigorously to cure it at once, if curable or worth curing; if incurable or not worth curing, kill it and dispose of the cadaver.

When the calf reaches breeding age, mate heifers with healthy bulls grown in the same manner. Before breeding, cleanse the genitalia of both sexes as carefully as practicable.

(2) When metritis exists and causes sterility, abortion, premature birth, or retained afterbirth, cure the metritis, cure it promptly and well, or send the cow to the butcher. Examine the genitalia of all suspicious cows often enough to keep track of the pathological conditions present. If the disease of the genitalia (ovaries, oviducts, or uterus) is incurable, slaughter the cow; if curable, cure her. Do not permit the herd bull to serve a cow which can not at that time conceive. Copulation intensifies the infection in the cow and imperils the health of the bull.

(3) Protect the bull by douching the external genitalia regularly before and after service.

(4) Do not introduce into the herd, except when absolutely necessary, new animals of either sex which may bring into the herd a more virulent strain of infection than that already present. In other words, keep no dangerously infected cattle of either sex or of any age in the herd. If they become diseased, cure them promptly or kill them as a menace to the herd.

A PRELIMINARY STUDY OF THE PATHOLOGY AND BACTERIOLOGY OF OVARITIS IN CATTLE

C. P. Fitch
Department of Pathology and Bacteriology.

The diseases of the genital organs of cattle are of exceedingly great importance because sterility often results and renders an otherwise valuable animal of little worth except for meat. Diseases of the ovaries have been recognized for a considerable period as of great importance among cattle. Hess, in his report in 1906, pointed out the close connection between cystic degeneration of the ovary, sterility and nymphomania. Albrechtsen, Williams and others have likewise studied the diseases of the ovaries and their relation to sterility and abortion. The work of these men, however, has been chiefly clinical and based largely on macroscopic findings. This study was undertaken in cooperation with Dr. W. L. Williams of the Department of Obstetrics and the Diseases of Breeding Cattle, to determine the microscopic as well as the macroscopic changes in ovaritis of cattle and also the micro-organisms which may be associated with these changes.

In human medicine many studies have been made of the tissue changes in ovaritis of women. The literature of this subject is so voluminous that no attempt will be made to complete a bibliography. MacCarty and Sistrunk in their article on Benign and Malignant Ovarian Cysts state: "An examination of the literature on the subject of ovarian tumors, especially ovarian cysts, shows that over 1,000 articles have been published since 1903. In the light of our present general knowledge of the subject, a review of such a wealth of recorded observations would be only of historical interest."

The pathological changes most often seen in ovaries, whether human or cattle, are different forms of cystic degeneration. Carcinomas and sarcomas are occasionally found as well as different varieties of mixed tumors. Malignant ovarian tumors in cattle are very rare and have no practical significance in the production of sterility. (In Dr. Williams' collection of over 3,000 diseased ovaries there are no carcinomas or sarcomas.) The question which has been agitated among human pathologists for many years and

which does not seem to be settled is, from what source does the ovarian cyst arise? Theoretically, according to Adami:

"Cystadenomas may arise from the epithelium of follicles, from the corpus luteum, from the superficial germinal epithelium, from certain tubules of the paroöphoron (Waldeyer), from displaced "rests" of the ciliated tubal epithelium (Kassmann), from remains of the Wolffian body (Kölliker's Markstränge). Attempts have been made to assign a particular origin to the cysts according to the character of the contained fluid, but this is a small point to decide upon, since, as is well known, the fluid varies considerably in different parts even of the same growth. As much depends upon absorption as upon secretion. Again, differentiation has been made on the ground of the presence or absence of papillary outgrowths and of ciliated epithelium. As Orth points out, however, it is difficult to draw a hard and fast line between the simple and the papillary cystadenomas, inasmuch as all sorts of transitional forms have been met with. It has been shown, moreover, that under certain circumstances non-ciliated epithelium may acquire cilia, and in man the existence of ciliated germinal epithelium has been proved. This being the case, it will readily be seen how difficult it is to come to any satisfactory conclusion as to the etiology of these cysts. A developmental origin for many of them is supported by several facts."

McCallum classifies cystadenomata of the ovary as follows:

"1. Simple ovarian cysts — so-called hydrops folliculi.
2. Pseudomucinous cystadenomata.
3. Serous cystadenomata.

"The division is not important since it is evident that it is based on no essential difference. The first type has long been supposed to arise from the Graafian follicles through mere accumulation of fluid in their cavities, and this view was supported by the finding of ova in the walls of the cysts (Rokitansky and others). Although rigorously upheld by Pfannensteil, it has been practically abandoned by most writers since the work of Nagel, v. Kahlden, and others, who have shown that these cysts are not derived from Graafian follicles, but from ingrowths of the germinal epithelium of the surface of the ovary. v. Kahlden traced this clearly in many cases and showed that the ova seen by several investigators were really protoplasmic masses somewhat resembling ova, but produced by the epithelial cells, perhaps as a futile effort on the part of those cells to carry out the function for which they were originally intended.

"The cystadenomata are also derived from solid or tubular ingrowths of the superficial germinal epithelium, and not from

the Graafian follicles nor from the so-called Pflüger's cords, which are groups of ova and epithelial cells. They are frequently single, but often arise from both ovaries simultaneously and are formed of one large cyst or of a great number of smaller ones (simple and multiocular cystomata)."

Mallory, in his discussion of diseases of the ovary, says:

"Cysts.— Several varieties of cysts occur in the ovary. Two arise in connection with new-growths. Three are due to distension of pre-existing cavities and will be considered before the others.

1. The first variety of retention cyst is due to dilatation of gland-like cavities which are probably remains of the Wolffian duct. Many of the cells are ciliated.

2. The second variety is due to distension of ovarian follicles and is lined with non-ciliated epithelium.

3. The third results from softening and an accumulation of fluid in the center of a corpus luteum or fibrosum.

"Tumors.— The epithelial tumors of the ovary are the most frequent and important. They are divided into three varieties according to their type of growth.

1. The *adenocystoma* is composed of glands and cysts lined with cylindric epithelium and filled with a thin to thick fluid containing pseudomucin. The tumors may attain a very large size and are usually benign.

2. *The papillary adenocystoma* is more likely to contain one cyst than many, does not attain a very large size and is filled with a thin, serous fluid. Papillary outgrowths may project from the wall at only one spot, or line the whole inner wall and fill the lumen of the one or more cysts present. Papillary growths may also occur on the outside of the tumor and even mask the cyst formation. The epithelium lining the cysts and papillary projections is tall, cylindric and often ciliated. The stroma may be slight or abundant. This variety of tumor may give rise to metastases in the peritoneal cavity and must be regarded with greater suspicion than the adenocystoma."

Kitt, in his Pathologische Anatomie der Haustiere, II Band, describes the following changes as occurring in the ovary:

"The developing, unruptured Graafian follicles give the ovary an abnormal cystic appearance. The theory that the follicles do not burst and develop into large cysts still remains to be proven. The failure of development of lutein cells due to some mechanical nervous influence is corrected by crushing the ovary. (Zschokke.)

"Simple or multiple enlarged cystic Graafian follicles are found most often in swine, next in horses and cattle, even in calves. The cysts vary in size from that of a walnut to an egg. They are cov-

ered with a smooth, fibrous, thin-walled capsule. Their contents are clear, watery and colorless (*Hydrops follicularis ovarii, Hydrocystis follicularis, Hydrocystoma*). These cysts are often in such masses that the ovary appears like a bunch of grapes. The fibrous stroma of the ovary becomes atrophied. Single, simple cysts may become as large as a cocoanut and may contain a bloody mixture, brownish in color, which acquires a sticky or clay-like consistency (*Hydrocystis haemorrhagica*).

" Cysts may develop within the ovary so that no change appears on the surface. The cysts are first recognized on sectioning the enlarged organs. They are surrounded by a zone of parenchyma 2 to 6 mm. thick. (*Hydrops follicularis parenchymatosa.*)

" Graafian follicles over 1½ cm. in diameter should be regarded as pathological cysts since egg cells are never found in them (Rubeli).

" Cystomas or Adenocystomas of the ovaries are generative tumors resulting from active proliferation of the epithelial tubules and cell columns which constitute the epithelial elements of the follicle and associated with a proliferation of the stroma. In cattle and horses especially one or both ovaries may develop tumors of enormous size, 20 to 30 or even 90 kilos in weight. They equal in size the stomach or rumen. The ovary is entirely transformed into the tumor, still the ovarian covering can be recognized on the outside of the tumor. The weight pulls the mesovarian down and the whole is found on the floor of the abdomen.

" The most common type of Cystoma is the *Adenoma cystoma cavernosum hemorrhagicum.* This is a hollow tumor, round or oval in form. It has a firm or hard consistency or may be slightly fluctuating. Externally it is covered with a tunica albuginea, a firm connective tissue coat several centimeters thick. The surface is smooth and slippery occasionally partially granular. From this connective tissue covering there project inward very abundant net-like interlaced cords which penetrate the center mass. This forms a mesh work with large or small cavities. The walls of these cysts are oval, either of a firm or gelatinous connective tissue. They vary in size from a hazel nut to a fist and are arranged in compartments. The contents are usually thin, watery or bloody fluid. Again, it may be thicker, of unmixed blood. It may be sticky or gluey, yellowish brown or dark brown, frequently fatty, cloudy or milky."

These four extracts are included as typical of the descriptions of cystic degeneration of the ovary included in a few of our best text books on pathology. They serve to illustrate also the variety of classifications used.

The ovaries which serve as the basis for this study have been collected from a number of sources. Some have been taken from cows which were known to be sterile and were slaughtered. Others have been taken from animals dead of various diseases. By far the larger number of ovaries have been taken from animals on the killing floor of a slaughter house. About seventy-five ovaries have been examined up to date. They were taken from animals of all ages, from two days to six or eight years. The macroscopic changes were noted in all and portions were fixed in Zenker's fiuid or 10 per cent formalin for microscopic examination. These were finally embedded in paraffin or celoidin, cut as thin as possible, the sections stained in hematoxylin and eosin and picrofuchsin was also used. A great deal of trouble was experienced in getting thin sections because the tissues were so hard.

In this preliminary paper a detailed description of the individual specimens will not be given. These will be included in reports to be made later. A general description of the material follows:

The average normal ovary of the cow measures $2\frac{1}{2}$ — 4 cm. by 2–3 cm. The ovary of a new-born calf is 1–1.5 cm. by 0.5–0.75 cm. They are irregularly oval in shape and the consistency is solid except where ripe Graafian follicles or cysts may be present. Corpora lutea are denoted by the characteristic color. Normally, they are slightly raised above the surface of the ovary and are spherical in form. Their average diameter is 1–2 cm.

According to Rubeli as quoted by Kitt, any Graafian follicle which is over $1\frac{1}{2}$ cm. in diameter should be considered a "pathological cyst." This is rather large based on the examination of our specimens. We have found cysts not exceeding 1 cm. in diameter which on microscopic examination of properly stained sections showed marked degeneration and desquamation of the wall, denoting that the Graafian follicle, if such it were in the beginning, was undergoing pathological changes.

The cysts found in the ovaries have been of various sizes up to 15 cm. in diameter. We have observed cysts in Dr. Williams' collection much larger than this. Sometimes but a single cyst occurred in an ovary. Often, however, several have been found giving the organs the so-called multiple cystic appearance. In these latter cases the ovarian tissue had pretty largely disappeared.

It could usually be made out at an edge of one of the cysts denoted by a thin band of tissue containing a few small Graafian follicles.

Deep or central cysts ranging in size from 1½–2 cm. in diameter have been found. These often are not revealed by visible changes on the outside but the ovary is usually more spherical. They are often discovered only when the ovary is incised. Often distinct remains of lutein tissue can be made out at the edge of these cysts. The contents are often turbid or yellowish in color. A corpus luteum which projects from the surface of the ovary 0.5–1 cm. and ranges in diameter from 1–2 cm. is occasionally found. The surface has but a very thin covering of epithelium. Often on incision this corpus luteum contains a cyst.

The contents of the cysts are usually a colorless, thin, watery fluid. It contains numerous fine particles which miscroscopically seem to be tissue débris. The fluid will coagulate somewhat on heating. As mentioned above, those cysts which occur within corpora lutea have a darker and more viscid content. This pigment is probably derived partly from the lutein cells and partly from the blood.

The microscopic examination of sections taken from ovaries shows that the majority of the cysts found in the ovaries of cattle are the so-called simple cysts. These may be single or multiple. The outer wall of a simple cyst is of connective tissue. The inner surface is made up of several layers of small round or slightly oval cells. The inner border is often irregular due to the difference in number of layers of cells. The contents are a clear fluid. Occasionally a small hemorrhage occurs in the wall of such a cyst. "When a Graafian follicle ceases to be a follicle and becomes a simple cyst of this description is not known. That such cysts originate from Graafian follicles is to be strongly suspected, because they have a similar lining and contain apparently the same clear fluid." (McCarty and Sistrunk.) We believe from our observations of the simple cysts of the ovaries of cattle that they arise from Graafian follicles in accordance with the view just quoted in relation to human ovaries. The follicles fail to rupture, due to some action, we believe, possibly of lutein cells remaining after the rupture of a previous ovisac. Many simple cysts are found in ovaries which have a persistent or unabsorbed corpus luteum. Occasionally a simply cyst is found in an ovary which

apparently does not have such a corpus luteum but if a careful examination is made of the whole ovary we have in these cases been able to find unabsorbed lutein cells. Clinical records show also that if a cow is sterile and palpation reveals a protruding or deep corpus luteum, if this be squeezed out, in many of the cases not otherwise complicated (salpingitis or metritis) the animal will breed. Many such ovaries are cystic and the cysts are ruptured at the same time that the corpus luteum is squeezed out. In the examination of the sections of ovaries, so far secured, there seems to be no evidence to conclude that simple cysts arise from the invagination of the germinal epithelium.

The next most common cyst originates in the corpus luteum. This has more or less lutein tissue making up its wall. In some cases you find corpora lutea deep in the ovarian stroma which has undergone cystic degeneration. The cyst ranges in size from 2–4 cm. in diameter and is sometimes larger. In many instances it is difficult to make out the lutein cells in the wall, but a careful examination will usually reveal them. The contents of these cysts are darker in color, thicker and more highly albuminous than that of the simple cyst.

We have so far found but a single case of the so-called cystadenoma. This was of the papillomatous type. It did not seem to be definitely malignant. The epithelial cells covering the papilla were not ciliated. No carcinoma or dermoids have yet been found by us.

The bacteriological examination of the ovaries has been carried out according to the method of Rosenau and Davis with a few minor necessary modifications. In brief, the technic consists in first securing the ovary in a sterile condition. This is much more difficult than in the case of securing the ovaries of women when on the operating table. Several plans have been tried. The ovary was first secured with sterile forceps and then dropped in a wide-mouthed flask fitted with a rubber cap and containing sterile saline. This was abandoned because the saline penetrated the ovarian blood vessels, and if, as was very possible, some slight contamination existed on the outside of the ovary, it was distributed by the saline throughout the organ. Next the ovary was wrapped in gauze, soaked in 1–500 bichloride. This method is still being used and is giving fair results. It has, however, an

inherent defect in that it usually requires some handling of the ovary and oftentimes with hands that are far from sterile. We are now securing the ovary with sterile forceps, flaming carefully the outside and then dropping into a sterile dry flask. This latter method apparently gives the best results. It must be kept in mind that sometimes twenty-four hours or longer pass after the time the ovary is secured before cultures can be made.

Just before the ovary is to be cultured it is removed from the flask, flamed by passing through a bunsen flame, and by means of sterile scissors the part to be cultured is cut away and dropped immediately into the tissue crusher. This instrument is described by Rosenau and the description need not be repeated here. We have had better success using the crusher than the sterile air-chamber recommended by Rosenau.

After the ovary is crushed, 3–5 c.c. of sterile bouillon is added and then by means of a wide-mouthed pipette from ½–1 c.c. of this suspension of tissue is transferred to the culture media. The medium used is agar prepared from meat infusion bouillon to which have been added 1 per cent glucose and 5 per cent glycerine, the whole made — 0.5 acid to phenolphthalein. To this medium, which is placed in test tubes 25 cm. tall, there is added just at the time inoculation is to take place 10 per cent sterile cow's serum. The serum is added to the liquid agar which has been cooled to 40–42 C. and the two are thoroughly mixed. The suspension of ovarian tissue is mixed throughout the height of the medium by means of a pipette and the whole quickly cooled. Thus all degrees of relationship to oxygen are obtained in such a culture, the strict anærobes could grow in the bottom and the ærobes near the top or on the surface of the medium. The tubes are incubated at 37.5° C. for at least three weeks.

Before giving the results obtained in the bacteriological examination of the ovaries of cattle, a brief review of the work of Rosenau and Davis on the ovaries of women seems appropriate. So far they have cultured 65 ovaries.

"In three of the patients the condition was rather acute, and in these the streptococcus viridans was recovered twice and the gonococcus once. The other patients had chronic pelvic disorders. In ten cases the cultures remained sterile after a week's incubation.

In the remaining 52 cases in which the ovaries showed fibrous and cystic degeneration, streptococci were isolated 30 times, the number of colonies ranging from one to relatively few, usually in the depths of the ascites-dextrose agar, to countless numbers. They were present in pure culture in 8, and associated in the others with the Welch bacillus, a few staphylococci or colon bacilli. Welch bacilli were found in small numbers in 21, diphtheroid bacilli in 10, a few colonies of staphylococcus albus in 9, the gonococcus in 2, the colon bacillus in 3, and an anærobic streptothrix in 1.

"The associated abdominal conditions were carefully recorded in 56 of the histories. Fibromyoma of the uterus was found in 18 patients, and the ovaries from 15 patients gave positive cultures — the streptococcus viridans being isolated from 11, and the Welch bacillus from 8. Salpingitis was reported 11 times, and the ovarian cultures were positive from 9, streptococcus viridans in 8, Welch bacillus in 3, and gonococcus in 2. Chronic appendicitis occurred 11 times, and the streptococcus was recovered from 8 of the ovaries. Eight patients had a chronic cholocystitis, and 6 of the ovaries cultures contained the streptococcus viridans.

"From our cultural studies of 65 ovaries it would appear that the streptococcus viridans is the most common organism associated with the chronic degeneration of the ovaries, being found in approximately 50 per cent of the cases. The Welch bacillus was found in 33 per cent but is probably of little importance. Other organisms are apparently found rarely in the usual chronic degenerative changes of the ovary. While the gonococcus is undoubtedly a common cause of acute infections of the ovary it is not found in the more chronic conditions, and our results suggest that some of the chronic pelvic conditions which were formerly credited to the gonococcus may have resulted from non-ascending infections with the streptococcus viridans."

Up to the present time we are prepared to report on the cultures obtained from 15 ovaries. These were secured from various sources and showed a variety of conditions (cysts) present. As stated before, a detailed description of each case will not be given in this preliminary report. Two ovaries from pregnant cows have been cultured. These ovaries did not show cysts and the cultures

remained sterile after four weeks' incubation. One case, that of a young calf, deserves special attention. This animal was born diseased, as it could not rise to suck. A stimulant administered gave the desired effect and the calf was able to take nourishment. The breathing was very rapid. The calf died when two days old. Post-mortem examination showed a pneumonia of both lobes of the lungs and parenchymatous nephritis. Cultures made from the lungs, liver, spleen and kidneys remained sterile except those from the lung which were pure of *B. coli*. The ovaries were removed and cultured. In these an organism developed which agrees culturally and morphologically with *B. abortus*. We are still working with this bacterium to assure ourselves of its identity with the Bang organism.

In all cases when a cystic corpus luteum was cultured vigorous growth was obtained. Several varieties of bacteria have been found. One ovary gave a pure culture of a long chained streptococcus. It was not of the *Strep. viridans* type. *B. coli* has been found in several ovaries. *Micrococcus pyogenes* has been found twice. A rod which seems to be a slight gas producer has been found three times. This organism has not yet been positively identified.

The work is being continued and more complete results will be reported at some later time. From the data so far obtained the following tentative conclusions seem justified.

1. Cystic degeneration of the ovaries of cattle is common. The character of the cysts is often *simple* but cystic corpora lutea or "*hemorrhagic cysts*" are found. Adenocystoma and papillomatous and carcinomatous cysts are relatively uncommon.

2. Cultures made from cystic ovaries of cattle show a variety of organisms to be associated with this condition.

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FURTHER REPORT ON THE DIAGNOSIS OF OPEN CASES OF TUBERCULOSIS

D. H. Udall, Department of Medicine

R. R. Birch, Superintendent Veterinary Experiment Station

One of the most interesting and important achievements of sanitation is that of being able to recognize the manner in which contagion is preserved and transmitted. In the suppression of a disease like tuberculosis, insidious and chronic in its course, and almost universally distributed, two fundamental factors must be clearly recognized. The first of these is the extent to which open cases may be diagnosed; the second is a knowledge of its direct and indirect channels of distribution. If, by any or all methods of diagnosis, we have a practical means of identifying every spreader or potential spreader among tubercular cattle, the method of control should be reasonably simple and certain. If, with all our practical methods of diagnosis, there still escape active or potential spreaders, the difficulty of control is greatly increased. In the suppression of most contagions more or less energy is exerted in an effort to meet unknown methods of transmission; but in the suppression of tuberculosis this feature has received little consideration; suppressive measures have been directed against known cases of infection. The protection of healthy animals against unknown spreaders has been neglected.

The experimental work here described has been conducted for the purpose of demonstrating the failures no less than the accomplishments of diagnostic methods for the recognition of open cases of the disease, or "spreaders." Each method of diagnosis has its value, and each scheme for the suppression of the disease possesses merit; all fail occasionally, while others fail as a rule. To successfully combat the disease it is highly desirable that we have exact knowledge of the relative value of each method of diagnosis, and that we should know the conditions under which failure may occur. The greatest obstacle to the suppression of tuberculosis is disregard of unknown cases of infection.

Previous to the discovery of tuberculin in 1898 a physical examination was the only known method of recognizing the disease. The names of Dr. Law in America, and of Siedamgrotzky in Sax-

ony, are mentioned as pioneers in this phase of the work. Both called attention to the results that might be expected from the removal of all physical cases, on the theory that this would remove all open cases or spreaders. Reports of successes then attained must be interpreted in the light of conditions then existing. The occurrence of the disease and opportunity for spread of the infection through traffic was much less in those days. Reports of the methods used are very meagre. So far as we know nothing is said about stabling conditions, conditions of feeding and watering, the handling of new-born and growing animals, or the exchange of stock. Our information on what may be termed individual segregation is limited. To judge from the relics of pioneer dairying found beyond the outskirts of modern dairy districts it is reasonable to assume that the methods of handling cattle under more primitive conditions provided greater protection against the spread of tuberculosis than those introduced with the era of fresh air, sunlight, cleanliness, and economy in care. Without detracting from the importance of all these improvements it is well to recall that dependence on their efficiency in the control of tuberculosis has resulted in a false sense of security and contributed to a marked increase in the spread of the disease. Exchange of the older custom of individual segregation for atmospheric improvement has not been counterbalanced by an increasing knowledge of the disease. It is a case where a little scientific knowledge has displaced a safe custom by a dangerous method. In the light of this interpretation the claims of the pioneers in physical examination are entitled to consideration and credit. A more recent recital of these claims, combined with a popular reception of the so-called physical diagnosis method, has led to its revival, and to the rather general belief that in the weapon of our forefathers is to be found relief from the increasing burden of this intolerable skeleton in the closet.

Having in mind the rather general impression among cattle owners and the general public that the spreaders can be recognized by a physical examination, the writers have striven to eliminate the infection from a herd kept under average dairy conditions. Like all experimental work it has carried us into other fields, and brought unexpected and surprising results. The prin-

ciple here involved is that of eliminating the chronic contagion, tuberculosis, from a herd without the removal of all cows known to be tubercular. With individual feeding and watering this method might possibly be successful, but in the so-called "modern" stable the opportunity for direct infection between cows is a severe test for any system.

A small experimental herd was established in 1911. The results of the first four years of work with this herd were reported in detail in the Annual Report of the New York State Veterinary College for 1914. Briefly summarized they are as follows to January, 1917, a period of six years.

In March, *1911,* nine reactors and six non-reactors were brought together where they associated closely, ate from a common manger, and drank from a common watering trough. In general both groups were an inferior lot, though none were included that were suspected of having symptoms of tuberculosis. A tuberculin test in December was positive to all in the tubercular group, and negative to the six others.

One tubercular animal was removed during the year: No. 20, a three-year-old, removed July 11 after six weeks in the herd, because of rapid loss in condition after calving, and death from tuberculosis of a guinea-pig inoculated with saliva from the throat. Three weeks later she died of generalized tuberculosis. No. 1 coughed occasionally, and No. 25 had an enlarged atlantal lymph gland; neither were removed. The tabulations for 1911 show fewer cattle, since those removed for other purposes early in 1912 have not been included.

In *1912* sixteen reactors and fifteen non-reactors were together for various periods. They were kept in good condition in comfortable quarters, and our earlier non-reactors were gradually replaced with calves. A tuberculin test in August was negative to six of the non-tubercular group, and nine out of fourteen of the tubercular. No new reactors were found.

During the year four cows were removed because of physical symptoms:

1. A nine-year-old, eighteen months in the herd, removed October 12 because of poor condition, history of cough in 1911, and

rales over the lungs. Autopsy revealed extensive pulmonary tuberculosis.

41. A three-year-old, thirteen months in the herd, removed July 18 because of poor condition and dyspnea. Autopsy: miliary and generalized tuberculosis.

42. A fifteen-year-old, thirteen months in the herd, removed July 18 because of poor condition, slight weak cough, and enlargement of a submaxillary lymph gland. Autopsy: a few lung lesions, evidently closed. Metritis after calving may explain the physical condition.

25. A three-year-old, seven and one-half months in the herd, removed because of an enlarged atlantal lymph gland. No autopsy. No. 1 should have been removed in 1911 when the occasional cough was observed.

In *1913* the physical examination was supplemented with guinea-pig inoculations with saliva taken from the esophagus; thus our original plan of removal based entirely on a physical examination was modified. The year was started with ten in the tubercular group from 1912, and eleven non-reactors, including the new-born calves. Tuberculin tests in May and November gave four negative reactions from the tubercular group and five positive reactions from the non-tubercular group — numbers 52, 70, 71, 74 and 58.

During the year seven tubercular animals were removed because of physical symptoms:

No. 11.—An 8-year-old cow, 21 months in the herd; removed January 3 because of poor condition; slight cough and rales. Autopsy — Extensive pulmonary tuberculosis.

68.— An 11-year-old, 5 months in herd; removed January 12 because of poor condition, and rales over both lungs. Autopsy — Tubercular lesion the size of a chestnut found in one lung. Sputum samples taken from the esophagus and trachea before death caused the death of guinea pigs from tuberculosis. This appeared to be a bad spreader regardless of the small size of the lesion. Emphysema of the lungs explained the presence of rales.

67.— A 13-year-old cow, 8 months in herd; removed April 12 because of poor condition, rales over both lungs and dyspnea on

exercise. Sputum positive to rabbit. Autopsy — Extensive pulmonary tuberculosis.

70.— A 4-year-old, 11 months in herd; removed August 9, three months after reacting, because of poor condition; cough, induced cough and nasal discharge. Bronchial secretions negative to guinea pig. Autopsy — Slight tuberculosis of a mediastinal lymph gland, and left lung. This was a recent infection with tuberculosis combined with a rather marked bronchial catarrh, possibly tubercular, though the very abundant bronchial secretion was negative to repeated pig inoculations.

62.— A 12-year-old cow, 13 months in herd; removed September 11 because of poor condition; cough, cough easily induced and very loud vesicular murmur. Autopsy — Generalized tuberculosis, open lesions in both lungs. Numerous samples of sputum from the esophagus killed guinea pigs in about two weeks.

65.— A 5-year-old cow, 14 months in herd; best physical condition of any cow in the herd. Removed October 16 because sputum from the esophagus caused the death of a guinea pig. Autopsy — Very extensive generalized tuberculosis.

66.— Aged cow, 14 months in herd; removed because sputum was positive to pigs; poor condition and cough. Autopsy — Extensive tuberculosis of the right lung.

This was a disastrous year. The significance of an occasional cough, induced cough and old age were underestimated. The poor condition was, in each case, credited to old age or metritis following abortion. Five of the seven were probably spreaders, while 62 and 66 were especially virulent.

In *1914* the year was started with eight reactors, and seven non-reactors, with two additional calves during the first quarter. A tuberculin test in November gave two negative reactions from the tubercular group and two positive reactions from the non-tubercular group. Five cows were removed because of physical condition, or positive sputum.

52.— A 2-year-old, born and raised in the herd, removed March 18 because sputum from the esophagus caused tuberculosis in a guinea pig. The left precrural lymph gland was enlarged. Autopsy — Extensive tuberculosis of both lungs.

29.— A 7-year-old cow, three and one-fourth years in herd: removed November 18 because of slight cough; cough induced at times, slight rales, indurated udder, sputum negative to pigs. Autopsy — A caseated mass about the size of a man's fist in one lung, numerous small tubercles had broken on the surface of the mucosa. One bronchial tube about one-half inch in diameter was filled with a tenacious mass for six inches. The tenacity of this mass may explain our failure to get tubercle bacilli in the samples.

64.— A 10-year-old cow, two years in herd; removed November 18 because of indurated swelling in right front quarter. Sputum found positive about two months after removal from herd. Autopsy — Tubercular mass six inches in diameter in one lung; this communicated with the bronchi and discharged thin pus. Udder tubercular.

74.— A 2-year-old removed November 18, two years from birth in herd, because of poor condition, induced cough and rales. Autopsy — Tubercular pleuritis, tubercles in the lungs.

51.— Eighteen-months-old, born and raised in herd; removed November 18 because of poor condition and suspicious submaxillary lymph gland. Autopsy six months later — Abscess between reticulum and diaphragm due to baling wire; this possibly accounts for the poor condition. Tubercular mass three inches in diameter in one lung; mediastinal, bronchial and right subparotid lymph glands slightly tubercular.

The year closed with five reactors and seven non-reactors on hand. The herd now consisted, with one exception, of young cattle born and raised on the farm; every animal was in excellent physical condition. Our previous failures were attributed in part to the old animals, and we were now confident that with a thorough cleansing and disinfection of the stable we could begin with this young herd and eliminate all infection by means of the combined sputum and physical examination methods.

1915 started with five reactors and eleven non-reactors. An ophthalmic test in July was negative to the non-tubercular group, and one reactor (3). A subcutaneous test in November gave one suspicious reactor in the non-tubercular group (92), and one negative (61) in the tubercular group. All of the tubercular group

reacted to either the ophthalmic or subcutaneous test during the year.

Two tubercular cows were removed November 22 for diagnosis practicums at the college; they were free from suspicion when removed, but neither has been returned.

3.— Eleven-year-old, five years in the herd. This cow was the last of the original herd of reactors. She was a pure bred Jersey and had never shown any signs of tuberculosis; she had been a very irregular reactor, and when posted July 18 was in poor condition. Autopsy — One small tubercle in a mediastinal lymph gland.

58.— Five-year-old cow, three and one-half years in herd, removed to college for practicums November 22 and never returned because of poor condition until the following June. During the summer of 1916 this cow regained her usual good condition. In December, 1916, she had rales, a cough, and induced cough on pinching the throat, or precussion of the thorax.

The year closed with three reactors, one suspicious, ten non-reactors, and four new-born calves. Our belief that the infection was practically eliminated seemed justified.

1916 started with four reactors, and 14 non-reactors. A composite sputum sample taken from the tubercular group February 14 was negative. A tuberculin test of the three reactors November 24 gave one suspicious, one negative and one positive result; thus our tubercular group contained the usual high percentage of non-reactors. In the other group the suspicious reactor (92) of 1915 became positive, and two more were added (100, 127) and one more (110) was suspicious. With the possible exception of 127 all new infections may be explained by direct contact with a spreader.

Three animals were removed:

85.— A 3-year-old, born and raised in the herd; reactor for two years; removed January, 1917, because too ugly to examine. Physical condition apparently excellent. Autopsy — Numerous tubercles in the mediastinal and bronchial lymph glands; tubercular mass about one inch by three-fourths inch on the lateral surface of the posterior lobe of the right lung. It is possible that this lesion communicated with a bronchus.

127.— Born in July, 1915, reacted in November, 1916. Removed January, 1916, because her condition was below that of others, not eating normally, very loud catchy inspiration. Autopsy — Negative.

73.— A 4-year-old non-reactor. Removed January, 1917, because her condition was below that of others, and slight glandular swellings with slight edema were found around the submaxillary glands. Autopsy.— Slight edema of the subcutis and among the salivary glands; salivary glands slightly enlarged and firmer than normal; lymph glands of head were slightly larger and lighter in color than normal.

The following tabulations show the record of the two groups for a six-year period.

NON-TUBERCULAR GROUP

YEAR OF BIRTH	Number	Dam	Age	Reacted	Years as non-reactor	Condemned	Years in herd	Years in herd reactors	Years in herd non-reactors
1911	28	.....	1.5	.....	.....	.....	.....	.....	6
1912	51	.....	April.....	Nov., 1914	2	Nov., 1914	2	.....	.....
1912	52	.....	April.....	May, 1913	1	Mar., 1914	2	.....	.....
1912	58	.....	3	Nov., 1913	1	Nov., 1915	3	.....	.....
1912	70	.....	3	May, 1913	0.75	Aug., 1913	1	.....	.....
1912	71	68	August...	Nov., 1913	1.25	.....	.....	4.5	.....
1912	73*	28	September	.....	.....	.....	.....	.....	4.25
1912	74	3	November	Nov., 1913	.....	.....	.....	.....	1
1912	75	11	November	.....	.....	.....	.....	.....	4.25
1913	83	.....	June.....	.....	.....	.....	.....	.....	3.5
1913	85	29	July.....	Nov., 1914	1.25	.....	.....	3.5	.....
1914	92	.....	February.	Nov., 1915	1.75	.....	.....	3	.....
1914	94	.....	April.....	.....	.....	.....	.....	.....	1.66
1914	99	74	June.....	.....	.....	.....	.....	.....	2.5
1914	100	51	June.....	Nov., 1916	2.5	.....	.....	2.5	.....
1914	101	28	August...	.....	.....	.....	.....	.....	2.33
1914	103	.....	September	.....	.....	.....	.....	.....	2.25
1915	110	.....	February.	Nov., 1916	1.8	.....	.....	2.0	.....
1915	113	.....	March....	.....	.....	.....	.....	.....	1.75
1915	127	83	July.....	Nov., 1916	1.32	.....	.....	1.4	.....
1916	130	61	September	.....	.....	.....	.....	.....	1.25
1916	137	.....	February.	.....	.....	.....	.....	.....	1

*Condemned December, 1916, because of edema and swollen lymph glands in the submaxillary region. Autopsy: No evidence of tuberculosis.
Month in age column indicates date of birth from 1912 to 1916. e. g. No. 71 was born in August, 1912. Refer to left-hand column for year of birth.

REACTORS

AGE	1911	Removed	1912	Removed	1913	Removed	1914	Removed	1915	Removed	1916	Removed	SYMPTOMS							AUTOPSY				TEST		
													Sputum	Poor cond.	Cough	Ind. Cough	Rales	Dyspnea	Udder	Lungs	General	Lymph gl.	Udder	Spreader	Tb. / test	Tb. — test
8	1	.....	1	Oct.	...	.....	...	.....	...	.....	...	.....	....	/	/	....	/	....	....	//	....	....	....	/	3	....
7	3	.....	3	.....	3	.....	3	.....	3	Nov.	....	.....	—	/	/	....	/	....	....	....	/	/—	....	....	3	3
6	11	.....	11	.....	11	Jan.	....	.....	....	.....	....	.....	....	/	/	/	....	....	....	//	....	....	....	/	3	....
3	20	July	....	.....	...	.....	....	.....	....	.....	....	.....	/	/	....	....	....	....	....	....	/	....	....	/	1	....
4	29	.....	29	.....	29	.....	29	Nov.	....	.....	....	.....	—	....	/	/	/	....	/	/	/	....	/	/	5	1
2	41	.....	41	July	....	.....	....	.....	....	.....	....	.....	....	/	/	....	....	/	....	....	/	....	....	/	1	....
15	42	.....	42	July	....	.....	....	.....	....	.....	....	.....	....	/	/	....	....	/	....	/—	/	....	....	/	1	1
1.5	.....	.....	61	.....	61	.....	61	.....	61	.....	61	.....	....	/	/	/	....	/	....	//	....	....	....	/	2	1
Old	.....	.....	62	.....	62	Sept.	....	.....	....	.....	....	.....	....	/	/	/	....	....	....	//	....	....	....	/	2	2
8	.....	.....	64	.....	64	.....	64	Nov.	....	.....	....	.....	....	/	/	....	....	....	/	//	....	....	/	/	2	....
4	.....	.....	65	.....	65	Oct.	....	.....	....	.....	....	.....	....	/	....	....	....	....	....	//	/	....	/	/	2	....
Old	.....	.....	66	.....	66	Oct.	....	.....	....	.....	....	.....	....	/	/	....	/	....	....	//	....	....	....	/	2	....
12	.....	.....	67	.....	67	April	....	.....	....	.....	....	.....	....	/	/	/	/	/	....	//	....	....	....	/	....	2
10	.....	.....	68	.....	68	Jan.	....	.....	....	.....	....	.....	....	/	/	/	....	/	....	/—	....	....	....	/	1	....
1912	.....	April	....	.....	52	.....	52	Mar.	....	.....	....	.....	....	/	....	....	....	....	....	//	....	....	....	/	2	....
4	.....	.....	....	.....	58	.....	58	.....	58	Nov.	....	.....	....	....	....	....	....	....	....	....	....	....	....	....	2	....
4	.....	.....	....	.....	70	Aug.	....	.....	....	.....	....	.....	—	/	/	/	....	....	....	/—	....	....	....	—	1	....
1912	.....	Aug.	....	.....	71	.....	71	.....	71	.....	71	.....	....	....	....	....	....	....	....	....	....	....	....	....	3	1
1912	.....	Nov.	....	.....	74	.....	74	Nov.	....	.....	....	.....	....	/	/	....	/	....	....	/—	....	....	....	/	2	....
1912	.....	April	....	.....	....	.....	51	Nov.	....	.....	....	.....	....	/	....	....	....	....	....	//	....	....	....	/	1	....
1913	.....	July	....	.....	....	.....	85	.....	85	.....	85	Dec.	....	....	....	....	....	....	....	/	....	/	....	....	3	....
1914	.....	Feb.	....	.....	....	.....	....	.....	92	.....	92	.....	....	....	....	....	....	....	....	....	....	....	....	....	2	....
1914	.....	June	....	.....	....	.....	....	.....	....	.....	100	.....	....	....	....	....	....	....	....	....	....	....	....	....	1	....
1915	.....	Feb.	....	.....	....	.....	....	.....	....	.....	110?	.....	....	....	....	....	....	....	....	....	....	....	....	....	?	....
1913	.....	July	....	.....	....	.....	....	.....	....	.....	127	Dec.	....	/	....	....	....	....	....	0	0	0	0	—	1	....

Age column indicates age, or date of birth of reacting animal.
Year columns show reactors with removals and recruits for each year.
/ = plus. — = minus. /— = slight.
All above parallel line are original reactors; all below are recruits from the non-tubercular group. Five in 1913, 2 in 1914, 1 in 1915, 3 in 1916. Twenty removals, eleven recruits, five tubercular remain.

Additional information on the diagnostic limitations of the physical examination method has been obtained through pig inoculations with sputum from cows reacting to tuberculin. The first series included 250 cows in five herds condemned by the tuberculin test, and segregated for the production of calves and pasteurized milk. They were held under a physical examination system subject to removal when symptoms developed. Fewer spreaders were found in the herds subject to the more rigid examination, but the percentage here was surprisingly high. In all inoculations guinea pigs of our own raising were used. We examined all cows at the time of taking samples. The minimum per cent of spreaders in any single herd was 10; the average for all, 20. Of the 50 spreaders identified by the sputum cup, 12 gave pulmonary symptoms (cough, induced cough, rales), 13 gave symptoms other than pulmonary, and in 25 no symptoms were found.

A second series of experiments with the sputum cup included cows that had passed both a physical examination and a tuberculin test. This work has been chiefly in tuberculin tested herds where the number of reactors at each test was unusually high.

Herd No. 1.— Certified milk herd of 54 cows; composite inoculation June 4 of nine pigs; on autopsy July 18 two tubercular pigs were found. Autopsy of the individual pigs September 1 revealed two positive and one suspicious case of tuberculosis among the pigs. A subsequent tuberculin test of the herd added one of the positive cases to the list of reactors.

Herd No. 2.— A tuberculin tested certified milk herd of about 150 cows. A change in the personnel of the testers was followed by a surprising increase in the number of reacting animals. A subsequent test at the end of three months revealed several more reactors, and a third test at the end of another three months revealed about fifteen more. Sputum samples of the last group were taken at the time of slaughter. Pulmonary lesions of the autopsied cows were slight or absent with one exception. The sputum sample from this cow was the only one to give positive inoculation results. She stood at the end of a common manger where water for the others had entered. It is highly probable that in this manner all the others of this group were infected, and that the disease was in the incubation period at the previous tests. The animals in this herd were kept under a very rigid physical

examination system; they were under the constant supervision of a competent veterinarian, and very few herds in the country would pass a better physical examination. The positive case passed the two previous tuberculin tests, but reacted at the third.

Herd No. 3.— A composite sample of 93 cows with a record of having passed the tuberculin test. Nineteen pigs were inoculated in composites of five to six cows per pig. Six of these pigs showed lesions of well-marked generalized tuberculosis on autopsy. Because of lack of time smears were not made to identify acid fast organisms. Individual inoculations from the positive composites have revealed five positive cases, and several suspicious ones on which the examination is not yet complete. It is quite obvious that the tuberculin test of this herd was faked.

Herd No. 4.— This herd comprises about 100 animals. It has been tuberculin tested for a number of years and all reacting animals removed. Purchases and sales were frequent. The number of reacting animals at each test was unusually high, and in January, 1915, the test revealed seven reactors and eight suspects. Individual sputum samples from this lot gave one spreader (37). This cow was bought on a test in 1912; she passed a second test in 1912, and two in 1913. At the usual test in 1914 the test was omitted because of recent calving. On autopsy a lesion communicating with the bronchi was found in one lung.

In March, 1915, five pigs were inoculated in composites of eight cows per pig; this included forty cows that had passed the tuberculin test in January. The pig inoculated with composite sample No. V was positive. Individual inoculations from this group were positive in two pigs, and an eye tuberculin test of the group gave four reactors, not including one of the spreaders; thus five of the eight were tubercular. Of the spreaders No. 19 had been tested semi-annually since 1908 without any rise in temperature; she failed to react to the eye test, and had never stood by the side of a suspect or reactor. To avoid any possibility of error three pigs were inoculated with a fresh sputum sample, and three with a two-months-old sample; all developed tuberculosis. The cow was in excellent physical condition. An autopsy in August revealed generalized tuberculosis, with one lung lesion about the size of a hen's egg. The second positive pig was inoculated with sputum from cow 49. The lesions of the first inoculation were suspicious,

and a sub-inoculation from these lesions developed a marked infection. At a tuberculin test in 1914 this cow gave a rise of 1 degree; in January, 1915, 0.8 of a degree. Autopsy December, 1915, revealed generalized tuberculosis.

Of the cows reacting in January, two — 35 and 37 — were generalized, and to these the non-reacting spreaders, 19 and 49, should be added. A study of the relative positions in the stable of the reactors and spreaders found in 1915 is suggestive of the manner of spreading. The cow stable accommodates about 60 animals; they are arranged in two rows with two common mangers for each row, about 15 cows per manger. With one exception all the reactors and suspects in this table had stood in sections with 35, 37, 19 and 49. The only reactor that had not stood in one of these sections had a record of a reaction as a yearling, when the rise in temperature was attributed to dysentery. Six reacting and suspicious yearlings had been segregated from birth and fed on pasteurized milk. Their infection was not easy to explain until a watering trough was found in the fence that separated the yard of the young stock from that of the cows. The cow stable was fitted with individual watering cups.

Composite tests of this herd have been made at intervals during the year 1916. In addition numerous individual samples have been received. Two individual samples have proved positive. Cow 47, born in 1910, has been segregated as a valuable, pure-bred reactor for several years. Her sputum was negative in January, 1915, but a sample received in April, 1916, proved positive. There is no report of any loss in physical condition, which was excellent when last seen a few months previous. Autopsy in November revealed a generalized extensive tuberculosis, including the udder. Thus our usual experience that a tubercular animal will become a spreader if kept long enough is supported by additional evidence.

The second tubercular pig in 1916 developed from sputum received in April and taken from bull 48. Correspondence in November states that he has developed a bad cough. An autopsy shortly afterwards revealed local tuberculosis of the retropharyngeal and mesenteric lymph glands. This suggests the possibility of finding tubercle bacilli in the esophagus in the absence of pulmonary lesions. We have evidence that this does occur when the

pulmonary lesions are slight, and have enough to suggest its possibility in animals with no recognized pulmonary lesions.

SUMMARY — EXPERIMENTAL HERD

Animals used	37	....	....	....	....	
Original reactors bought	....	14	....	....	....	
Reactors recruited from non-tubercular group	....	10	....	....	....	
Reactors in herd	....	....	....	4	....	
Reactors condemned	....	....	20	....	....	= 54 per cent.
Non-reactors in herd	....	....	....	13	....	
Non-reactors condemned	....	....	1	....	....	
ositive to sputum-cup test	....	....	....	....	8	= 21.6 per cent.
Open pulmonary tuberculosis found on autopsy	....	....	....	....	15	= 77.2 per cent.

Time in herd of original tubercular animals before being condemned.			Years in herd of non-reactors of recruits to tubercular group.		Age of condemned tubercular animals recruited from non-tubercular group.	
Age	No.		No.		No.	
3	20	0 yr. 4 mo. out.	51	2 yr. 0 mo.	51	2 yr.
8	1	1 yr. 6 mo. out.	52	1 yr. 0 mo.	52	2 yr.
6	11	1 yr. 9 mo. out.	58	1 yr. 0 mo.	58	3 yr.
4	29	3 yr. 8 mo. out.	70	0 yr. 9 mo.	70	1 yr.
2	41	1 yr. 4 mo. out.	71	1 yr. 3 mo.	127	1 yr. 4 mo.
15	42	1 yr. 4 mo. out.	85	1 yr. 3 mo.		
1.5	61	4 yr. 6 mo. in.	92	1 yr. 9 mo.		
Old	62	1 yr. 2 mo. out.	100	2 yr. 5 mo.		
8	64	2 yr. 4 mo. out.	127	1 yr. 4 mo.		
4	65	1 yr. 3 mo. out.				
Old	66	1 yr. 3 mo. out.				
12	67	0 yr. 9 mo. out.				
10	68	0 yr. 6 mo. out.				
7	3	4 yr. 8 mo. out.				

CONCLUSIONS

Several significant facts are emphasized by this summary:

1. About 50 per cent of the herd have been condemned and about one-third of the remaining 17 are tubercular.

2. A large percentage of tubercular animals were condemned within two years after admission on a physical examination basis, or as new reactors from the non-tubercular group; 50 per cent of the latter were condemned within two years after their first reaction.

3. Susceptible animals acquire the infection in a comparatively short time after exposure. Reactors in the non-tubercular group were first discovered at an average of one and one-half years, so that the period of incubation must have been considerably less.

Tubercular recruits from animals born into the herd acquire the disease most readily when young. This indicates that young animals are more susceptible to the infection, and supports the theory held in human medicine, that most infection is acquired in youth.

4. A large per cent of the tubercular animals developed into open pulmonary cases. Fifteen out of nineteen gave either sputum-cup evidence, or post-mortem evidence of being spreaders. Pig inoculation results showed that small lung lesions were eliminating freely into the bronchi, while inoculations of sputum from cows having large distinctly open lesions were sometimes negative; this suggests that all pulmonary lesions should be regarded as open. These facts have been observed repeatedly in sputum-cup work in other tubercular and certified herds. If seems evident that most tubercular animals become spreaders if kept long enough, and that many of them reach this condition in a short time. We have posted only one tubercular cow in this experiment in which the lesions were undoubtedly closed and healed. A further significant fact is our failure to find pulmonary symptoms in 75 per cent of cows whose sputum caused tuberculosis in pigs; though a few cases which gave very distinct pulmonary symptoms were negative to the sputum-cup test.

5. A comparison of the different methods of diagnosis shows that all are far from perfect. The value of each is purely relative and increases when used in combination with other methods.

Our chart of reactors shows 44 positives and 14 negatives in known tubercular animals. An analysis of the negatives reveals one closed and healed, four open and extensive, one open and slight, two still in herd. We have reason to believe that all recent infections gave positive reactions. After 1914 tests were made annually. One negative reaction in a tubercular animal occurred in 1915; and excluding new reactors, two of three tubercular animals failed to react in 1916.

6. The real direct value of the sputum cup is yet to be established; it is evident that the cow must become a spreader before recognition by this method is possible. Considered individually it is valuable as a means of detecting open cases in non-reactors, indicating spreaders among cattle believed to be the object of fraudulent testing, and protecting free herds against the introduc-

tion of spreaders. Contrary to what seems to be a prevailing belief, sputum-cup examinations are neither complicated or difficult to make. As previously noted, it will indicate many spreaders passed over by a physical examination, and a few that escape both a physical examination and a tuberculin test. Indirectly it should be of value in emphasizing unknown spreaders, and the necessity of individual segregation within the herd.

7. It is evident that reliance upon a physical examination alone for the suppression of tuberculosis in our experimental herd kept under usual conditions of feeding and watering, is a failure. The extent to which some individuals have developed lesions before they were recognized is enough to weaken one's confidence in his own judgment and powers of observation, while the development of symptoms resembling those of tuberculosis, but due to other causes, leaves one in a state of confusion. This is especially true if the final opinion is to be checked by autopsy. Without attempting to pass judgment on the value of a general examination of dairy cattle or the value of this method in the suppression of tuberculosis in general, we have convincing evidence that in our hands it reveals only a small percentage of open pulmonary cases. There are many reasons why a physical examination of dairy cows should be practiced, and why it should be extended, but in company with all other diagnostic methods its limitations must be recognized as determined by experimental and practical tests, and not by a confession of faith or public opinion. Employed alone it reduces the number of spreaders in a badly infected herd, but it cannot be made to remove all, or nearly all. It is of great value in detecting the occasional non-reacting spreader, and as a means of eliminating udder lesions from a herd that produces market milk. The physical examination of large animals is hard work. In a sense the meaning of the term is entirely relative, depending on the experience, judgment and application of the examiner. For general use in dairy inspection a uniform standardized system should be established in order to define the minimum requirements of an examination and to indicate how often it should be repeated.

8. In infected herds control depends on a knowledge of the cows that are not tubercular fully as much as a knowledge of those that may be found to be tubercular. In large herds where the

infection is well distributed, and in herds where exchange is frequent, it may be assumed that a knowledge of all the cases of open tuberculosis that appear in the herd is practically impossible. So long as tubercular animals continue to be found in a herd it is a safe assumption that unrecognized cases remain there. In estimating the value of different methods of diagnosis too much emphasis has been placed on their achievements, and not enough on their failures. This has resulted in placing too much reliance on the value of a diagnosis in suppressive schemes.

Our results emphasize the following well-known quotation from Professor Bang: "Owners of cattle ought to prevent the contamination of calves and other animals still healthy." With the discovery of tuberculin and the knowledge it brought of the wide prevalence of tuberculosis numerous plans for the protection of healthy animals were devised. They were all based on the assumption that the diagnosis of tuberculosis had become an exact science. Great emphasis was placed on the restrictive influence of ideal atmospheric conditions, otherwise termed the suppression of tuberculosis by improved and salubrious conditions of life, a phrase evidently borrowed from human hygienists, and an expression of an excellent idea. The uniform human custom of eating from individual dishes, and drinking from individual cups, was not emphasized. To provide for a clean sweep of light, air, and cleanliness, stables were modernized by stripping them of every unnecessary device, and the individual cow manger has now been replaced by what may now be conservatively termed a cross between a sewer and a gutter. Daily washing and disinfection can make it nothing else. It is repulsive to every principle of good hygiene. From a bacteriological standpoint higher and lower animals stand on a common platform, and what individual would relish eating and drinking from a soup bowl common to fifteen other individuals, some of whom are known to have open pulmonary tubercular lesions? It is time to stop fooling the farmer with sunshine and devote more time to the rigid details of investigation.

LEUKEMIA AND PSEUDO LEUKEMIA IN THE COMMON FOWL

E. M. PICKENS

Department of Pathology, Bacteriology and Meat Inspection

Leukemia is a primary disease of the blood and blood-forming organs. It is in the majority of instances a chronic disease characterized by the presence of an enormous number of leucocytes in the circulating blood, associated with anemia, though the essential characteristic is not so much the large number of leucocytes as the varieties and proportions of these which are found (Burnett).

Two varieties of leukemia are described, (1) the lymphatic (lymphoid, lymphemia), and (2) mixed celled (myeloid, myelogenous, etc.), which are differentiated by the condition of the blood and the blood-forming organs.

The histological changes in lymphatic leukemia and pseudo leukemia are very similar. In fact, many investigators consider them different stages of the same disease. Most cases of pseudo leukemia, however, run a chronic course and show no disposition to change to leukemia. The essential difference between the two diseases is found in the blood. Very slight or no changes at all are present in the circulating blood in pseudo leukemia, while an enormous increase of lymphocytes is found in leukemia.

These two conditions with the allied forms make a very important group of maladies in the lower animals, especially in the common fowl. This importance is due, not so much to the economic loss caused by them, as to their relation to these affections in the human subject, in which they generally prove fatal. It necessarily follows here, as in most other diseases, that where the mortality is high, the condition is but little understood, and treatment in a scientific manner cannot be applied. Happily, the chicken with its apparent susceptibility, and the readiness with which it may be obtained, lends itself most admirably to experimental work.

During the past five years, a number of cases of leukemia and pseudo leukemia of the fowl have come under our observation, and in the following paper a summary of the literature together with

a brief presentation of the clinical and anatomical aspects of the disease will be given.

Historical

Probable cases of leukemia were observed and commented upon from the beginning of the 19th century by Buchart (1801), Androl (1823), Hodgkin (1832), Donne (1830), and many others, and the disease was imperfectly recognized in current text books (Piony, Velpeau, Rokitansky) in which it was regarded as an obscure "suppurative hematis;" Donne (1839) found, in a case at autopsy, that the blood cells were more than one-half "white or mucous globules," and attributed the cause to a failure of transformation of the leucocytes into red cells.

The first steps in the elucidation of the malady were made when Craigie (1841) demonstrated an entire absence of suppurative foci in the body and concluded that the purulent material was absorbed from the enlarged spleen of which the histological structure does not favor the gathering of pus in abscesses.

The first good descriptions of the disease were given independently of each other in 1845, by Virchow in Berlin and Bennett in Edinburgh. Virchow named it Leukemia, meaning white blood. The connection between the affection of the spleen or lymph glands and the increase of the white blood corpuscles was early recognized by Virchow, that of the bone marrow was first established by Neumann in 1869.

Leisering (1858) was the first to recognize leukemia in animals. Great attention has been given it only in the last twenty years, so that at present quite a number of positive cases are known. Siedamgrotzky (1878), Johne (1879), Nocard (1880), Fröhner (1885), Wolff (1892), de Jong (1903) have carefully studied the disease.

The cases in fowls described by Butterfield (1905) and Jutaka Kon (1907) were undoubtedly leukemia, although the blood of the affected birds was not examined. Warthin (1907) seems to have been the first to recognize the malady and study the blood changes. Ellermann and Bang (1908) recognized and studied the disease as such. Valuable work relative to its clinical aspect was furnished by Hirschfeld and Jacoby (1907), also by Skiba (1909). Schmeisser (1915) was able to transmit leukemia through several generations.

OCCURRENCE

Leukemia has been observed in domestic animals in comparatively few cases. Nocard in 1880 cited 43 cases, 9 in horses, 5 in cattle, 4 in swine, 22 in dogs and 1 in a cat. In the Prussian army in 1890 to 1895 there were 26 horses reported as having leukemia. Other cases reported are mostly in dogs. Authentic cases in sheep, goats, rabbits and guinea pigs have not, so far as I am aware, been observed.

In poultry six spontaneous cases have been observed in this laboratory. Hutyra and Marek state, "The disease occurs exclusively among chickens, and among these in an enzootic and even an epizootic form." The malady has been observed in Denmark, Germany and in this country. Actual data as to its frequency do not seem to be at hand. It is doubtless a rare disease but is probably not so rare as the number of recorded cases would lead one to suppose.

Pseudo leukemia has been observed in horses, dogs, swine, calves, cows, cats and the domestic fowl. Hutyra and Marek state: "The disease occurs more frequently than leukemia, for which it is frequently mistaken." Leukemia in chickens is in about half of the cases also manifested by the symptoms of pseudo leukemia. In this laboratory I have had the opportunity to observe about thirty cases. Here again but little data as to the frequency of occurrence are available.

ETIOLOGY

The actual cause of leukemia in man, as well as the essential nature of the disease, remain obscure. Various affections (malaria, syphilis, rickets, etc.) have been regarded as predisposing causes. The same is true of pregnancy, lactation, traumatism, exposure and other influences. To some heredity has seemed to be an important element. In these and other cases the lesions are considered to be a simple hyperplasia.

The conception of leukemia as a neoplasm, fathered by Banti and others, is conceded by many workers as being the true explanation. The formation of metastases, the infiltrative and destructive growth, the failure of inoculation and transplantation, etc., all favor this view.

However, infection is believed by many pathologists to be the direct cause of leukemia, and various forms of bacteria have been described as being its cause. There are certainly some very striking facts in favor of its infectious nature the most important being the apparent contagiousness in a few cases, the similarity of the acute form to an infectious disease, and the similarity of certain conditions produced by bacteria such as tuberculosis. The various micro-organisms, believed to be its cause, need not be enumerated, as none of them have been proved to be pathogenic. Bodies resembling protozoa (Aurers bodies) have been found in the blood and the lymph glands, but the nature and significance of these are uncertain.

In the fowl Hutyra and Marek state that the disease is produced by some kind of an infectious substance, the nature of which is still unknown. Only this much is known from the investigations of Ellermann and Bang, that the cell-free filtrate from organ-emulsions may transmit the disease and therefore the etiological role of an ultra-microscopic organism does not appear to be excluded. Hirschfeld and Jacoby were able to transmit it but their results were negative with Berkefelt filtrates of organ-emulsions from leukemic chickens. Burchhardt, and again Schmeisser, transmitted the disease with organ-emulsions, not filtered.

The virus seems to be present in all affected organs, the spleen, liver, bone marrow, etc. The infectious properties of the organs, however, are lost in a few days after death. The bodies resembling protozoa seen by Kon were also found by Ellermann and Bang. These authors, however, refrain from expressing an opinion as to the nature of the bodies. Hirschfeld and Jacoby, on the other hand, found peculiar long bacilli which produced a temporary anemia with lymphocytosis in an experimental chicken; still they do not yet desire to consider this bacillus as the cause of the disease.

Pseudo leukemia appears to be as closely related to leukemia in the fowl as in man. The histological changes in each case are practically identical and many workers believe that they are either different stages or different types of the same disease. The theories of hyperplasia, neoplasm and infection all have their supporters. The consensus of opinion seems to be with the hyperplastic theory. In the fowl, if we accept the work of Ellermann and

Bang, we find that an inoculation of organ-emulsions from a leukemic fowl may produce either leukemia or pseudo leukemia in the injected bird.

CLASSIFICATION

A classification of leukemia and its allied forms which will be acceptable to all pathologists appears to be an impossibility at the present time. Until the controversy over the etiology of the disease has been settled, the followers of each theory, as given above (viz., hyperplasia, neoplasm, infection, etc.), will support a classification in harmony with their theory.

The followers of hyperplastic or metoplastic theories, as the case may be, put emphasis on the type and character of the cell, in so far as the type of the disease is concerned, whether the cells are lymphocytes or myelocytes. They place special emphasis on the character and location of the lesions. They generally exclude the conditions known as lymphosarcoma and small round cell sarcoma.

To many the idea of transplantation and growth of cells seems more plausible, although there is good evidence in favor of the idea of metaplasia. It appears then, that if we know accurately all the cellular types existing in the bone marrow and in the lymphoid tissue, which are the blood-forming organs concerned, and if we assume that each is capable of undergoing an independent hyperplasia, we shall be able to construct a tabulation of all the possible diseases arising in this way. Sternberg has made such a table which has been modified and improved by MacCallum. It is as follows:

A. Hyperplasia of lymphoid tissues:

a. With leukemic blood.

1. With swelling of lymphoid tissues and infiltration of organ — *Chronic lymphoid leukemia; acute lymphoid leukemia.*

2. With tumors originating in various situations and invading tissues — (*chloroma*), *Leucosarcoma; chloroleucosarcoma.*

b. Without leukemic blood.

3. With tumors involving bone marrow — *Lymphoid or plasma-cell myeloma.*

4. With general swelling of lymphoid tissue —*Pseudo leukemia.*

5. With regional invasive tumor-like growth — *Lymphosarcoma.*

6. With stigmata of general maldevelopment — *Status lymphaticus.*

B. Hyperplasia of myeloid tissue:

a. With leukemic blood.

7. With myeloid infiltration of organs — *Myeloid leukemia myeloblastic leukemia.*

8. With tumors of myeloid tissue — *Chloromyelosarcoma (myeloid chloroma).*

b. Without leukemic blood.

9. With tumors of myeloid tissue — *Myeloid myeloma.*

C. (Included though probably not related.) Tumor-like swelling of lymph glands with nodules in spleen, liver, lungs, etc., granulomatous alterations of lymphoid tissue of specific morphology, apparently infectious in origin — *Lymphogranulomatosis* or *Hodgkin's disease.*

To the supporters of the infectious theory, the classification becomes one of inflammations. Here we have the acute and the chronic forms of the diseases. Special attention is given to the character of the lesions and to the organs affected. The leukemic or aleukemic condition of the blood is also important. With this theory, as with hyperplasia, the sarcomas, lymphomas, myelomas, etc., are not included.

In the domestic animals the infectious theory is based upon very strong evidence, especially in poultry, where transmission experiments have been carried out repeatedly. Here Hutyra and Marek divide the diseases as follows:

A. Leukemia in mammals in which they designate the acute and chronic forms. They also differentiate the lymphatic and myeloid types.

B. Leukemia in chickens, which generally lasts eight to fourteen days, but may last one to three months. This would signify an acute and chronic form. The lymphatic and myeloid types of the disease are also mentioned.

C. Pseudo-leukemia, under which they include the following terms:

Hodgkin's disease, aleukemic lymphadenia, sublymphatic leukemia, lymphadenia and pseudo leukemia lymphatica.

In this article the term "leukemia" will be used in the restricted sense, and to designate the disease only when both the pathological findings of the blood-forming organs and of the blood itself warrant it. The term "pseudo leukemia" will also be used in the restricted sense, and will refer to the same condition found in leukemia minus the blood changes.

SYMPTOMS

The symptoms in leukemia and pseudo leukemia are practically identical with the exception of the absence of the marked pathological blood picture in pseudo leukemia. The period of incubation seems to be from one to two months.

The acute forms are generally very similar to the chronic. The different stages, of course, are not so sharply defined in the acute form. In fact, in some instances, no disease is detected. The bird appearing lively at the morning feeding may be found dead before night.

In the chronic form, especially, the disease may be ushered in with an anemia. This is first noticed in the comb and wattles. They become pale and perhaps icteric. The membranes of the head and the skin of the legs likewise show a paleness or icterus. Blood obtained by a needle prick may be of a pale red color. Stained preparations show polychromatophilic red blood cells varying from normal size to megalocytes. The red blood cells generally diminish from normal (3,000,000 to 4,000,000 per cu. mm.) to as low as 1,000,000 per cu. mm. Simultaneously a diminution of the

hemoglobin content (normal 60 to 70) is noted. It may drop to 15 per cent or 20 per cent. A rather profuse diarrhœa has been noted, making its appearance simultaneously with the anemia. It may be present in the absence of the anemia and generally continues throughout the course of the disease. In several cases persistent hemorrhage occurred as a result of slight wounds on the comb and wattles.

The increase in leucocytes in leukemia generally takes place shortly after the onset of the anemia. Usually the increase is gradual although it may advance by leaps and bounds. The total number of leucocytes may increase from 30,000 (normal) to 100,000 or even 600,000 per cu. mm. The proportion of the white to the red corpuscles (normal 1 to 100 to 1 to 200) may fall to 1 to 2 or 1 to 3. At the same time the percentage of the different cells changes to such an extent that the mononuclears represent 60 to 90 per cent and the polynuclears and lymphocytes are forced into the background. In other cases the lymphocytes or the myelocytes may show the marked increase.

Throughout the course of the disease, the appetite generally remains good, especially in the chronic cases. In the acute it may become precarious and toward the end the bird may refuse food altogether.

In the acute cases, cyanosis has been noted at the end. In the chronic, the abdomen may become pendant. The birds generally die from an apparent toxemia or excessive emaciation.

Number and Sources of Specimens Studied

The specimens reported in this article were taken from twenty-two birds. In a few cases, a part of the bird only was available for examination. The most unfortunate condition, however, is in regard to the lack of study of the condition of the bone marrow. When the material from these cases was being collected, the bones were not available. The pathological findings described and the conclusions drawn are therefore made without a knowledge of the changes which had taken place in the bone marrow.

Of the twenty-two diseased birds referred to above, thirteen came from a poultry farm where from 1,500 to 2,500 birds have been kept. These cases were collected during the last five years,

six of them have been received within the past twelve months. Two of these were classed by their blood picture with the leukemias. The eleven remaining ones were aleukemic or pseudo leukemic. It was impossible to obtain any information in regard to the possibility of contact of these birds.

At another poultry farm a few miles distance from the first, four cases occurred. Here the deaths all took place within a period of two months. Of these two proved to be leukemia and the other two lacked the typical blood picture and hence were placed with the pseudo leukemias. At this farm the birds, about 125 in number, were all kept together. Another bit of information obtained was the fact that the stock from this farm originally came from the first.

The five remaining cases came from widely different parts of the State and no history of contact with other cases could be obtained. One of these was found to be leukemia, and the other four were pseudo leukemia.

NORMAL FOWL

The following data of the normal fowl have been compiled from a large number of birds. They are confined to those parts of the body which are involved in leukemia.

Blood. The blood of a fowl differs markedly in some respects from that of mammals. A brief description of its constituents together with normal counts will, therefore, be given.

The blood is thick in consistency, dark red in color and clots very rapidly.

Red cells. The red cells are elliptical disks measuring 11.5 x 6.5 to 13.5 x 8 microns. The nucleus is the same shape as the cell, staining a deep blue and slightly pyknotic. The cytoplasm is yellow and glassy.

Leucocytes. According to Burnett's classification five varieties of leucocytes are found in the circulating blood of the domestic fowl and are differentiated in preparations stained by Jenner's or Wright's stains by the following characters:

Variety I. Lymphocytes. This variety includes cells usually about the size of or smaller than the average red corpuscle. Each

has a relatively large nucleus that occupies nearly all of the cell. The nucleus is usually round, but may be incurved or show a deep notch or sinus at one side. The cell body usually shows as a narrow rim about the nucleus. Both nucleus and cell body are coarsely reticular. With careful staining a nucleolus may be seen. The cell body has a strong affinity for basic stains, often staining a deeper blue with Jenner's stain than the nucleus. With Wright's stain the cell body has a greenish blue while the nucleus has a dark violet tint. Cells falling into this group have practically the same appearance as in mammals.

Variety II. Large mononuclears. Cells belonging to this variety are larger than those of Variety I, usually about twice the diameter of the average red corpuscle. The nucleus usually occupies only about one-half of the cell and is situated at one side of the center. Its shape is oval or curved (kidney or horse shoe shape). Both nucleus and cell body are finely reticular and stain less deeply than do those of the lymphocytes. The cell body is faintly basophile. These cells have much the same appearance as in mammals.

Variety III. Polymorphonuclears (Polynuclears). The nucleus in this variety is several lobed, the different lobes being connected with slender portions. Rarely the nucleus consists of several separate parts. In shape it is polymorphous; it may be twisted, spirally coiled, S shaped, U shaped, Z shaped or elongated. It is usually well stained and is coarsely reticular. The cell body contains many large granules, spindle shaped with tapering ends, rod shaped with rounded ends, club shaped or oval, that stain a reddish color with Jenner's stain or eosin and methylene blue, and a dark reddish with Wright's stain. With Ehrlich's tri-acid stain they take a deep reddish purple color. The granules vary in size from one to three microns in length by about one micron or less in width. This cell has generally been classed as an eosinophile; but it evidently does not belong to that variety.

Variety IV. Eosinophiles. The nucleus is markedly polymorphous exhibiting the same shapes as in mammals. The cell bodies contain round eosinophilic granules, similar to the eosinophile found in mammalian blood. Except in the shape and size of the

granules, this cell corresponds to the polymorphonuclear leucocyte found in the blood of mammals. The nucleus exhibits the same shapes as in mammals. In affinity for stains, the granules resemble the polymorphs rather than the eosinophiles. The cell has active ameboid movement.

Variety V. Mast cells. In this variety the nucleus usually takes the stain so faintly that it is difficult to make out its shape. It varies in shape from rounded or curved to by-, tri- or many-lobed. The cell body contains many strongly basophile rounded or oval granules that take a deep violet tint with Jenner's and a royal purple with Wright's stains. Mast cells as a rule are slightly larger than the eosinophiles.

Thrombocytes. Thrombocytes in the blood of the domestic fowl are elliptical, oblong or spindle shaped cells with an elliptical to broadly oval nucleus. In size the cell has nearly the length and about one-half the width of the average red cell. The nucleus occupies about one-half the length, and nearly the entire width of the thrombocyte, and is usually situated in the central part of the cell. The cell body is pale and often contains one or more clear vacuoles, and occasionally one or more compact, rounded, deeply staining (deep purple with Wright's stain) bodies about the size of or somewhat larger than a mast cell granule. These bodies are probably a result of degeneration. The thrombocytes show a marked tendency to collect in clumps. In fresh blood, and in the less thinly spread parts of films, they collect in masses in which it is difficult to distinguish the outline of individual cells. This indistinctness of cell outline and structure shows another property of these cells, that is, their vulnerability. They change quickly when taken from the blood vessels, passing through a characteristic series of changes. Both cell body and nucleus become less distinct, the cell body losing its structure first. Finally both become structureless, appearing in stained preparations merely as a diffusely stained mass, the nucleus being distinguishable by having a slightly deeper stain.

The following tabulation gives a summary of examinations of the blood of normal domestic fowls by different investigators.

Red corpuscles per cu. mm.	Leucocytes per cu. mm.	Thrombocytes per cu. mm.	Hemaglobin, per cent	Size of red corpuscles	Authors
3,324,000	17,921	....	.76	....	Burnett
2,300,000	12–29,000	....	....	....	Warthin
3,637,000	20,081	....	....	....	Moore
3,017,000	33,777	55,272	87.3	13.1x8	Mack
3–4,000,000	35,000	60,000	60–70	....	Personal Observation

Number of Leucocytes and Percentages of Varieties in the Blood of Normal Fowls

Leucocytes per cu. mm.	Lymphs I	Large mono II	Polymorph III	Eosinophiles IV	Mast cells V	Author
33,777	54.9	6.2	32.7	2.7	3.3	Mack
12–29,000	35.5	14.5	21.5	10.0	2.0	Warthin
17,921	58.0	5.5	28.8	3.3	4.3	Burnett
35,000	55.5	8.2	30.8	4.3	1.0	Personal Observation

Gross anatomy. The gross anatomy has been taken largely, although not entirely, from Schmeisser. A fowl of average size and weight (1,760 grams) has abundant subcutaneous fat and large muscles. The inner surface of the skin is slightly yellow. The peritoneal cavity contains an omentum rich in fat. It originates from the anterior surface and ventral margin of the gizzard extending caudal with the intestines. The liver is a bi-lobed organ, weighing approximately 50 grams or 2.8 per cent of the body weight. It is of a reddish brown color, moderately soft and friable. The cut surface shows small blood vessels in longitudinal and cross-section.

The spleen measures 2 x 1.5 x 1 cm. and weighs one gram or 0.25 per cent of the body weight. It is rather soft and reddish brown. Beneath the capsule bluish white malpighian corpuscles, slightly larger than a pin point, may be distinctly seen. On section, the capsule is very delicate, the trabeculae are few, but usually definite, containing small blood vessels. The pulp is not

raised above the surface of the capsule. The kidneys weigh 12 grams or 0.7 per cent of the body weight. They are reddish brown and lobulated.

Histology. In the liver the lobular formation is not prominent. Periportal spaces are not easily made out. They contain the usual vessels, surrounded by very little fibrous tissue. Lymphocytes are present either scattered diffusely or in follicles. The liver cells are arranged in cords, separated by capillaries, which generally contain red cells almost exclusively. In the spleen the malpighian corpuscles are numerous and distinct. They are composed of the usual lymphocytes surrounding small arteries. Red blood cells are limited to the pulp, where they occur more or less in clusters, although sinuses or enclosures of any kind cannot be definitely demonstrated. The pulp does not differ materially from that of the liver or spleen. The kidneys have a structure similar to the human kidney.

METHOD OF EXAMINATION

In the study of these diseases, the routine given below was adhered to as closely as possible. The history was always obtained in as much detail as the owner of the bird was able to give. Unfortunately in several cases no history could be procured.

The external appearance of the bird was carefully noted, and if alive a blood count made when symptoms warranted it. The fowls were then placed in the animal house for observation. Here they were watched for several days and numerous blood counts made. At death a post mortem examination was made, in which each organ was carefully observed and the macroscopic changes noted. Smears of the heart blood were always prepared when possible and stained after both Wright's and Giemsa's methods. The organs were weighed, and pieces of tissue were taken for section. For the preservation of the specimens Kaiserling's fluid was used. Sections fixed in Zenker's and Orth's fluids were cut by the paraffin method, as thin as possible, never thicker than six microns. The specimens were stained after the following methods: Hemotoxylin and Eosin, Hemotoxylin and Picrofuchsin, Wright's, Jenner's, Giemsa's and Ziehl Neelsen's. Tuberculosis was always very carefully excluded.

Gross Anatomy

In both leukemia and pseudo leukemia the birds were generally greatly emaciated and presented a dejected appearance. The macroscopic and histological changes were practically identical in the two diseases with the exception of the blood picture. The organs affected in the order of occurrence seemed to be the liver, spleen, kidney and more rarely the ovary and omentum.

The liver has been affected more or less extensively in every case examined. It was generally enlarged, the size varying from a slight enlargement to an enormous organ occupying nearly all the space in a pendant abdomen and weighing as high as 237 grams. In this case the liver weight was 24 per cent of that of the fowl. The exterior of the organ was usually smooth and of a light mahogany color intensely mottled with greyish white areas, irregular in shape, with clean cut edges. These areas ranged in size from small points or streaks to as large as two centimeters in diameter. In a few cases the surface of the organ was literally covered with raised, rounded greyish white nodules, varying in size from those just visible to as large as one and one-half centimeters in diameter. In one case the liver showed large rounded raised areas. In several cases the capillaries of the capsule were injected. On section the capsule was generally found to be thin and tense. The liver tissue was often rather soft and friable, and in a few cases projected out beyond the capsule. The greyish areas of the surface were always found to extend throughout the depth of the organ. They were of the same shape and color as on the surface. No evidence of encapsulation was observed macroscopically. The liver tissue was always more or less congested and showed evidence of varying degrees of degeneration.

The spleen showed lesions in all but two cases. It was generally enlarged, weighing from two to fourteen grams. The surface of the organ was usually smooth and of a bluish brown color, intensely matted with grayish white irregular areas with clean cut borders. A few cases showed the surface of the organ to be literally covered with small yellowish white raised nodules similar to those described on the liver. On section, the capsule was found to be thin and taut. The splenic pulp was soft and generally projected out beyond the capsule. The greyish areas or nodules observed on its

surface were also found throughout its depth. More or less congestion was always present. The malpighian corpuscles could usually, although not always, be made out.

The kidneys were much enlarged in a few cases and in others were practically normal in size. Their color was a pale reddish-yellow and they were more or less mottled similar to the liver and spleen. The raised nodules on the surface of the liver and spleen were not observed on the kidney. On section the greyish areas were found scattered irregularly throughout the cortex and medulla. The kidney tissue was more or less degenerated and always appeared to be congested.

The ovary was affected in only one case observed. In this case, it was about the size of a normal non-functioning organ, greyish white in color and contained practically no eggs.

The omentum, like the ovary, was affected in only one case. In this case, it was literally covered with small greyish-white nodules varying much in size and shape. Many were just visible, while the larger ones were one and one-half centimeters in diameter. They were pedunculated, being attached to the omentum by short pedicles. Others were thin and nearly flat with rounded edges resembling fish scales. On section a rather heavy connective tissue capsule was observed. The pulp was yellowish-white and somewhat firm. Numerous blood vessels filled with blood were seen scattered through the nodules. No trabeculae could be detected.

HISTOLOGICAL FINDINGS

The liver was usually badly affected, from one-third to three-fourths of its tissue being replaced with lymphoid areas, which were found scattered throughout the organ. These areas varied in size from a collection of a few cells around a blood vessel to large tracts comprising several lobules. They appeared to originate in some cases in the inter-lobular tissue around the inter-lobular vein and extend into the lobules. In other cases, they seemed to take their origin from around the central vein within the lobule. Very rarely their origin appeared to be independent of blood vessels.

The areas were made up of lymphocytes which were closely packed into a fine connective tissue reticulum. The small cells were in every way similar to those normally found in the blood.

The others varied in size from only a slight enlargement to double that of the normal lymphocytes. Their nuclei were large, generally round, and occupied most of the cell body. They stained intensely and often diffusely. No granules were observed in these cells. The protoplasm was basophilic. Numerous mitotic figures were observed in these cells but never in the small lymphocytes. The edges of the lymphoid areas in several cases were clean cut while in others more or less diffuse. In one case numerous areas showed the presence of a distinct connective tissue capsule. This, as far as I have been able to find, has never before been reported.

The liver tissue, away from the lymphoid areas, generally showed more or less cloudy swelling and in a few cases fatty degeneration was also observed. The cords of liver cells, especially in the vicinity of the lymphoid areas, were often contracted and the capillaries between them were practically obliterated, showing evidence of pressure. In other cases the liver cells were large and appeared to be more or less oedematous. The capillaries were greatly distended and engorged with blood.

In the leukemic cases the large or small lymphocytes, as the case might be, were greatly increased in number in the blood vessels. The ratio of red cells to white varied from one to two to one to nine or ten. Numerous mitotic figures were observed in the large lymphocytes in both the large blood vessels and capillaries. No evidence of division was seen in the small lymphocytes.

The spleen showed numerous lymphoid areas scattered throughout the organ. These areas varied greatly in size and number in the different cases. Some showed only a few relatively small foci while others presented areas which occupied nearly the whole organ. They always appeared to originate from the malpighian corpuscles. Their structure was similar to that described in the liver. The cells were either the large or small lymphocytes or both and appeared to be identical with those found in the other organs. Division was often observed in the large lymphocytes in the lymphoid areas, and in case of leukemia, also in the blood vessels. These cells were generally, although not always, thickly crowded into a reticulum which was fine, but varied somewhat in different cases. The borders of the areas were similar to those

described in the other organs. One case, the one referred to in the description of the liver as possessing nodules which were encapsulated, also showed a capsule around many of the areas in the spleen. In a few cases the connective tissue of the spleen was greatly increased. This was noted principally as islands and extended to the walls of the arteries of the malpighian corpuscle. The splenic pulp away from the lymphoid areas was nearly normal. In several cases numerous red cells were observed scattered through the pulp. The blood vessels were similar to those described in the liver.

The kidney often showed similar lymphoid areas scattered throughout the cortex and medulla. These foci varied in size from a collection of a few cells to large tracts. They appeared to originate in the glomeruli in the cortex and around the small capillaries in the medulla. The areas showed the same structure and type of cell as found in the other organs of the case in question. The kidney tissue was generally degenerated. Cloudy swelling, fatty degeneration and sometimes necrosis and disintegration were observed. The blood vessels were usually engorged with blood and presented no characteristics not mentioned in the description of them under the other organs. In the ovary the functioning tissue was practically all replaced with a diffuse lymphoid tissue. The cells and structure was otherwise similar to the cases heretofore described. The supporting tissue was practically all of the organ that remained.

The mesenteric nodules showed a rather heavy connective tissue capsule within which lymphoid tissue was found. These nodules did not differ essentially in structure from the areas described in the other organs. A few trabeculae were observed which contained numerous injected blood vessels. The lymphoid cells were of the same type found in the other affected organs.

One case of leukemia and one of pseudo leukemia have been selected from the group to be described in detail as representative of the two diseases.

Leukemia. Fowl No. 3215. Single comb, white leghorn hen about one and one-half years old. This bird was brought to the laboratory with a history of not having been well for the past ten

days. It had eaten at times, but had not taken food for three days. Diarrhea had been present for some time. The membranes were of a dirty yellow color (icteric). The comb, wattles and legs were pale. The bird was somewhat emaciated and presented a generally unthrifty condition. It would stand for hours with its legs doubled up as if it were hovering chickens. It was placed in the animal house for observation. A blood count was made immediately, and thereafter regularly at periods of three to five days, until death, a little over two months later. The first blood count gave the following results:

Hemoglobin (Dare's)	40%
Red cells	2,500,000
Leucocytes	75,000
Small lymphocytes	10%
Large lymphocytes	65.4
Large mononuclears	6.1
Polynuclears	16.1
Eosinophiles	2%
Mast cells	0.4%

For the next six weeks the bird remained in about the same condition. The diarrhea continued unabated and the appetite was precarious. Emaciation was daily becoming more marked. The blood picture was typical of leukemia. It varied slightly from count to count, but continued in one general direction. A decrease in the number of red cells to 2,000,000 had taken place. The hemoglobin (Dare's) had fallen to 35 per cent. The total leucocytes were increased to 120,000, which were divided as follows: Small lymphocytes, 5 per cent; large lymphocytes, 79.4 per cent; large mononuclears, 5 per cent; polynuclears, 8 per cent; eosinophiles, 2.1 per cent, and mast cells, .5 per cent. The next two weeks, the bird declined very rapidly, refusing food nearly the entire time. She became so weak that she could stand but a minute or two at a time, and finally died from exhaustion. The blood count two days before death was typical of the findings at this stage of the disease and was as follows:

Hemoglobin (Dare's)	33%
Red cells	1,800,000
Leucocytes	120,000
Small lymphocytes	4.5%
Large lymphocytes	79%
Large mononuclears	4.0%
Polynuclears	10.5%
Eosinophiles	1.3%
Mast cells	0.7%

Post Mortem Examination. The bird was extremely emaciated and all the external organs were pale. The liver was not greatly enlarged. It weighed 65 grams. The left lobe measured 8 cm. long by 6 cm. wide and the right lobe measured 5 cm. long and 4 cm. wide, which is practically the normal size. The organ was of an amber or mahogany color, showing the usual mottled appearance. Its edges were slightly rounded, and the capsule tense. On section, the mottled appearance was found to extend through the depth of the organ. The consistency was soft and somewhat friable. The blood vessels were filled with blood. Evidence of degeneration was present.

The spleen was about double its normal size, and was of a bluish-grey color. It did not show the mottled appearance. On section, the pulp was found to be rather firm. The malpighian corpuscles could not be made out. The blood vessels were filled with blood. The other organs were practically normal.

Histological Examination. The liver was not badly affected. The lymphoid areas were relatively small and even restricted to practically the immediate vicinity of the inter-lobular veins. Only about 50 per cent of these were affected. The areas had the usual structure of fine stroma in which lay the lymphoid cells. They were not as thickly crowded as in most of the other specimens studied. The cells were of the large lymphocytic type, and numerous mitotic figures were observed. The edges of the areas were not sharply defined. The liver tissue appeared to be practically normal. Some degeneration was present and also some evidences of pressure. The capillaries between the cords of cells were greatly distended and were engorged with blood. The larger vessels were also filled with blood. The leucocytes were increased in number

in both the capillaries and the larger vessels. They appeared to be in a proportion of one leucocyte to seven or eight red cells.

The spleen was nearly normal. The stroma was slightly heavier than is usually found. The connective tissue of the malpighian corpuscles was also increased. The pulp contained enormous numbers of large lymphocytes. The blood vessels were congested and showed an increase in the number of leucocytes. Their proportion to the red cells appeared to be about the same as found in the liver. Mitosis was common.

Pseudo leukemia. Fowl No. 3399, single comb, white leghorn hen, two years old. This bird was brought to the laboratory with no history. It showed evidence of diarrhea but no anemia was present. The bird was in fair flesh. The blood count was normal.

Post Mortem Examination. The liver was much enlarged, weighing 105 grams. The left lobe was 11 cm. and the right lobe 9 cm. in length. The organ was of a light mahogany color and mottled. The tissue was soft and friable and the blood vessels were filled.

The spleen was somewhat enlarged, weighing six grams. It was nearly round and measured 3½ cm. in diameter. The malpighian corpuscles could be distinctly seen in places. The pulp was soft and congested. The mottled appearance was also present.

Histological Examination. The changes in the liver varied somewhat from the cases previously described. The areas of infiltration were sharply circumscribed and appeared to originate within the lobule and around the central vein. They were generally large and in many places had become confluent with their neighbors. In places, strands of liver cells still remained within the areas. The lymphoid tissue did not vary materially in structure from the other described. The stroma was somewhat finer than that observed in some cases. The cells were small lymphocytes. The liver tissue showed evidence of pressure. Cloudy swelling was present but not marked. The capillaries were unusually small and contained only a normal number of leucocytes. The blood picture in the larger vessels appeared to be normal.

In the spleen, the lymphoid areas were very numerous and of large size. In fact, they appeared to occupy about one-half of the organ. Their edges were sharply defined. In places, they seemed

to have shoved the splenic pulp aside, making it very dense. In parts of the organ where several of the areas were in close proximity, this crowding gave them the appearance of a lobulated tumor. They originated in the malpighian corpuscles and their structure was similar to those described previously. The stroma was fine and the cells were very thickly crowded into the spaces. They were of the same type as those described in the liver.

The splenic tissue was somewhat denser than normal. Many red blood cells were seen scattered through it. A few practically normal malpighian corpuscles were observed. The small blood vessels were generally empty while the large ones were filled with blood, which appeared to be normal.

Discussion of Cases. Of the cases examined, five proved to be leukemia with well marked blood changes. Two were probably pseudo leukemia with a slight increase in leucocytes; and the remainder, fourteen in number, were pseudo leukemia with no blood changes.

In the leukemic group, the proportion of red cells to leucocytes varied from 2 to 1 in one case, to 10 to 1 in another. The lesions, with the exception of the blood picture, appeared to be identical with those of pseudo leukemia. The cells were of the small lymphocytic type in three cases. In one they were the large lymphocytes and in another the small cells predominated, but a few of the large ones were present. The large cells often showed mitosis. The small ones were apparently normal lymphocytes.

In the two cases which were probably pseudo leukemia but contained a slight increase in leucocytes in the circulating blood, the tissue changes were not different from those already described. They were both of the small lymphocytic type.

In the third or pseudo leukemic group, four cases contained the large type of cell. The remaining cases, with one exception, contained small lymphocytes. This case presented both the large and the small cells, but the small variety predominated. One case showed a peculiar characteristic in that the lymphocytes in the center of the lymphoid areas were badly degenerated, giving the appearance of a necrotic center. Two other cases showed many degenerated lymphocytes scattered throughout the lymphoid areas. Another feature in one case was the connective tissue encapsulation of several of the lymphoid areas in the liver and spleen.

In all three groups, the tissue changes varied slightly from case to case, but the major changes were the same in both leukemia and pseudo leukemia, with the exception of the blood picture.

Course and Prognosis

The course and prognosis of leukemia and of pseudo leukemia in the common fowl appear to be practically identical, and if we accept the theory that they are etiologically identical, the similarity is easily explained. They both have an acute and chronic form. The acute varies, from the birds showing no symptoms and being found dead, to the diseases lasting for about eight to fourteen days. The chronic form may extend over a period of from one to three months and even longer. Unexpected deaths apparently take place at any time.

The prognosis in both cases is unusually grave. Ellermann and Bang observed two spontaneous recoveries, Hirschfeld and Jacoby one. With these exceptions, the diseases have nearly always proved fatal.

Diagnosis

The diagnosis of leukemia and pseudo leukemia presents two entirely different problems. In leukemia the symptoms of anemia, unthrifty condition, precarious appetite, pendant abdomen, etc., should be taken into consideration. They, however, mean but little as they may occur in several other diseases. In the living bird a careful examination of the blood is of the greatest importance. If the typical leukemic blood picture is present a positive diagnosis may be made. If the disease happens to be of the lymphatic type and the increase in lymphocytes is not marked, difficulty may be encountered in differentiating it from tuberculosis and fowl typhoid. Skiba states that he has observed a simple leucocytosis, which occurs very frequently in chickens, in which the increase in leucocytes reached such a degree that the relation of the red cells to the white was as low as 26 to 1. In certain of these cases the increase may be almost wholly in the lymphocytes. To complicate the situation still more, Ellermann and Bang have found by their inoculation experiments that only 50 per cent of the artificially infected birds show the leukemic blood changes. In these cases the only available method of diagnosis is by post

mortem and histological examinations. If the typical lymphoid areas are found in the blood forming organs, namely, liver, spleen, bone marrow, etc., a diagnosis of leukemia or pseudo leukemia may be made. There is, as far as I have been able to find, no method by which these two conditions may be absolutely differentiated in the absence of the leukemia blood picture.

In pseudo leukemia, the blood changes are absent. The only remaining methods of differentiation and diagnosis are the symptoms which are by no means pathognomonic and the post mortem findings. To these a bacteriological, as well as a histological, examination have to be added, the one for the exclusion of some of the chronic infectious diseases, and the other to identify the lymphoid areas in the liver, spleen or other organs.

TREATMENT

In fowls no known methods of treatment are available for either leukemia or pseudo leukemia. Ellermann and Bang observed recovery in one case after treatment with arsenic. It could not, however, be decided whether this did not represent an accidental spontaneous recovery. The Röntgen treatment proved unsatisfactory in another case. In man, for pseudo leukemia the best results seemed to be obtained with a systematic administration of iron, iodine and arsenical preparations. The Röntgen rays are also used. Generally speaking, the mortality of both leukemia and pseudo leukemia in fowls is 100 per cent.

CONCLUSION

From a study of twenty-two cases of leukemia and pseudo leukemia, together with a review of the literature on these diseases, the following conclusions seem to be warranted.

1. That leukemia and pseudo leukemia in the common fowl are not rare.

2. That the lymphoid areas generally originate in the liver from around the interlobular or the central vein; in the spleen from the malpighian corpuscles and in the kidney from the glomerulus in the cortex and from around the capillaries in the medulla.

3. That the large lymphocyte (a young proliferative cell, abnormal in the general circulation) may be present in the lesions of leukemia and pseudo leukemia.

4. That degenerated lymphoid cells and even necrotic centers may be present in the lymphoid areas.

5. That encapsulation of the lesions may occur.

6. That leukemia and pseudo leukemia in the common fowl are closely allied to those diseases in the species homo.

Acknowledgments. The writer wishes to acknowledge his indebtedness and express his thanks to Drs. V. A. Moore and S. H. Burnett, through whose valuable suggestions and assistance this work was made possible. Thanks are also due the Poultry Department of the New York State College of Agriculture for their hearty cooperation in supplying specimens.

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DESCRIPTION OF PLATES

Plate No. 1. Liver and spleen of case No. 3360. Small lymphocytic leukemia (actual size). Spleen and one lobe of liver enormously increased in size. The other lobe of the liver is slightly larger than normal. The organs were of a pale greyish red color and were not mottled.

Plate No. II. Right lobe of liver of Case No. 3443. Small lymphocytic pseudo leukemia (actual size). This organ was a mahogany red, mottled with greyish areas.

Plate No. III. Liver of Case No. 3496. Small lymphocytic pseudo leukemia (actual size). The lobulation was not present in this case. The mottled appearance was absent.

Plate No. IV. Intestines, gizzard and liver of Case No. 3318. Large lymphocytic pseudo leukemia (actual size). The lymphoid nodules may easily be seen on the serosa of the intestine, the mesentery, the gizzard and the liver which occupies the lower left hand portion of the picture.

Plate No. V. Kidneys and ovary of Case No. 3483. Small lymphocytic pseudo leukemia (actual size). The mottled appearance of the organs may be easily made out.

Plate No. VI. Lymphoid areas in the liver and spleen.

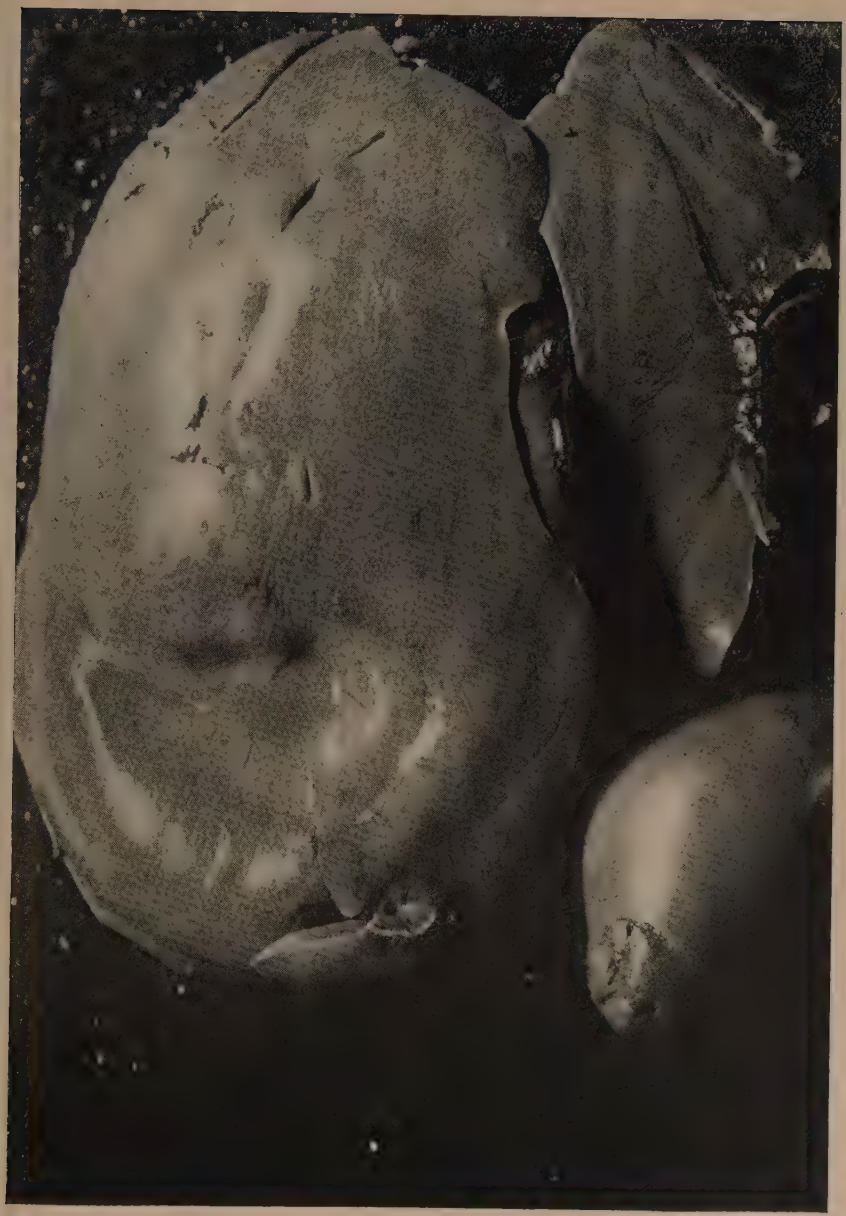
Fig. 1. Liver from Case No. 3482. Small lymphocytic pseudo leukemia (x 250). The lymphoid area occupies the central part of the picture.

Fig. 2. Spleen from Case No. 3518. Large lymphocytic pseudo leukemia (x 225). The lymphoid area occupies nearly the whole picture.

Plate No. VII. Lymphoid areas in the kidney and ovary.

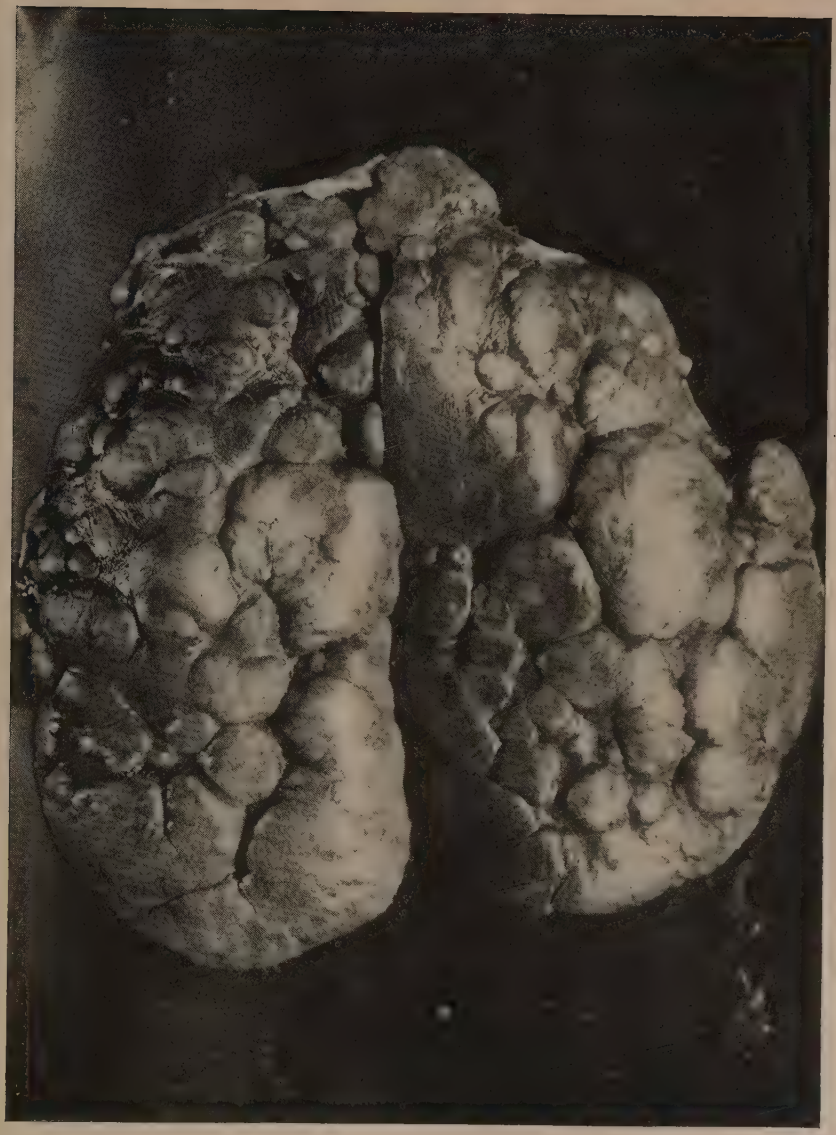
Fig. 1. Kidney from Case No. 3449. Large lymphocytic pseudo leukemia (x 250). A lymphoid area occupies the center of the picture. Remnants of glomeruli may be seen within the area.

PLATE I



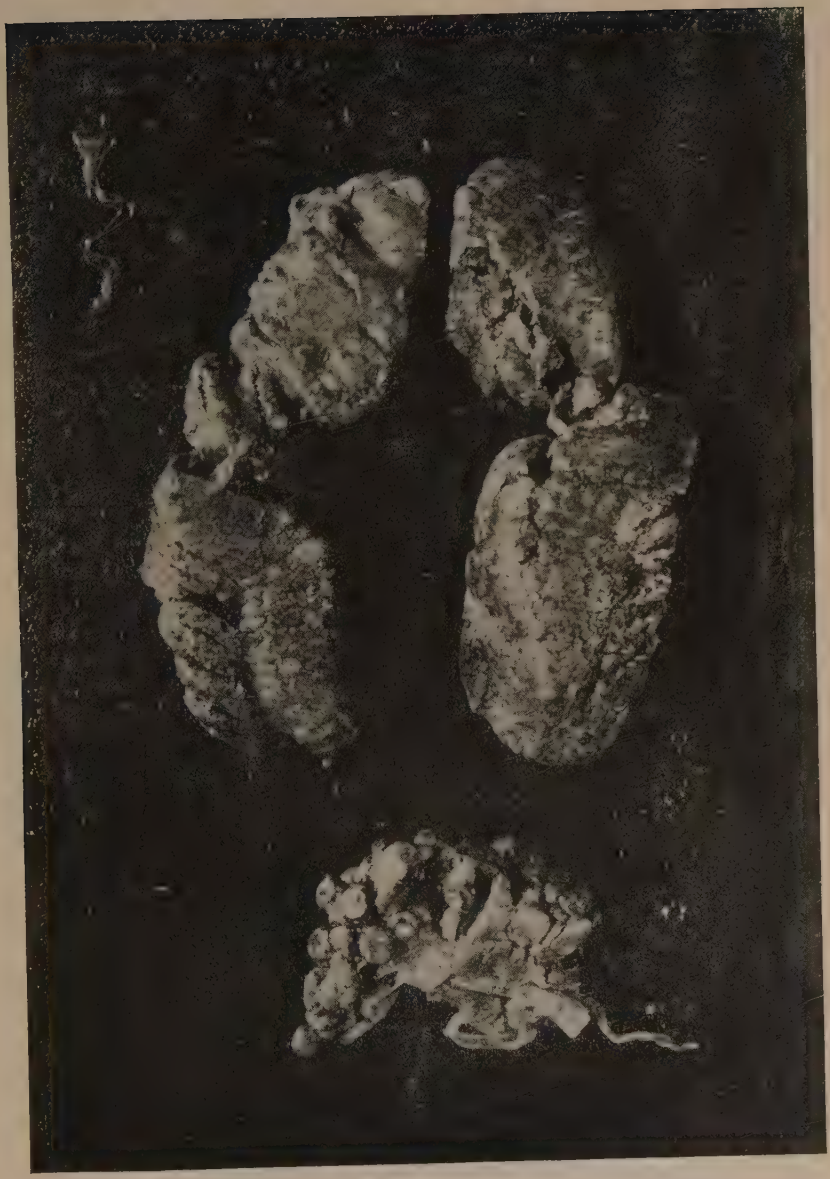
Liver and spleen, showing relative size of parts.

PLATE III



Liver, showing nodular surface.

PLATE V



Kidneys and ovary, showing lymphoid areas.

PLATE VI

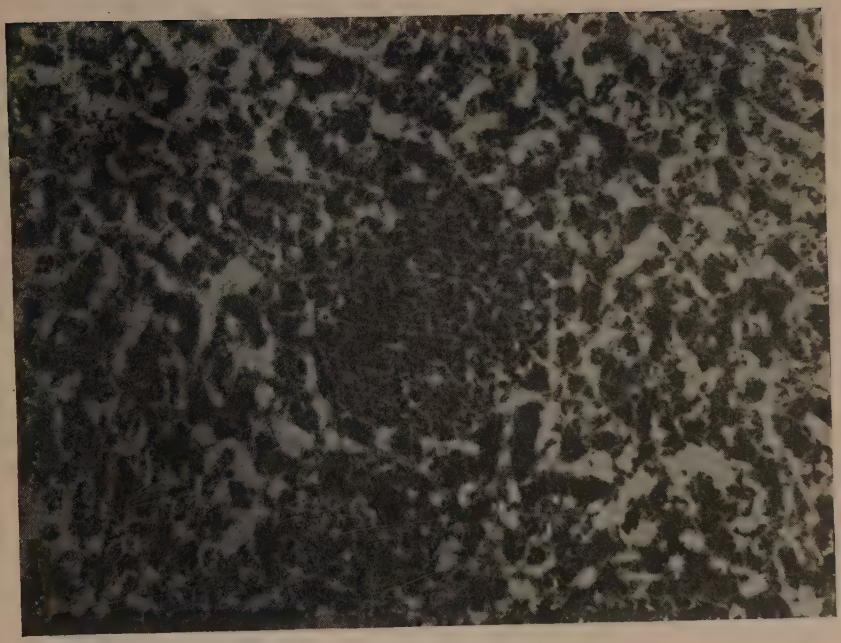


FIG. 1
Section of liver, showing lymphoid infiltration.

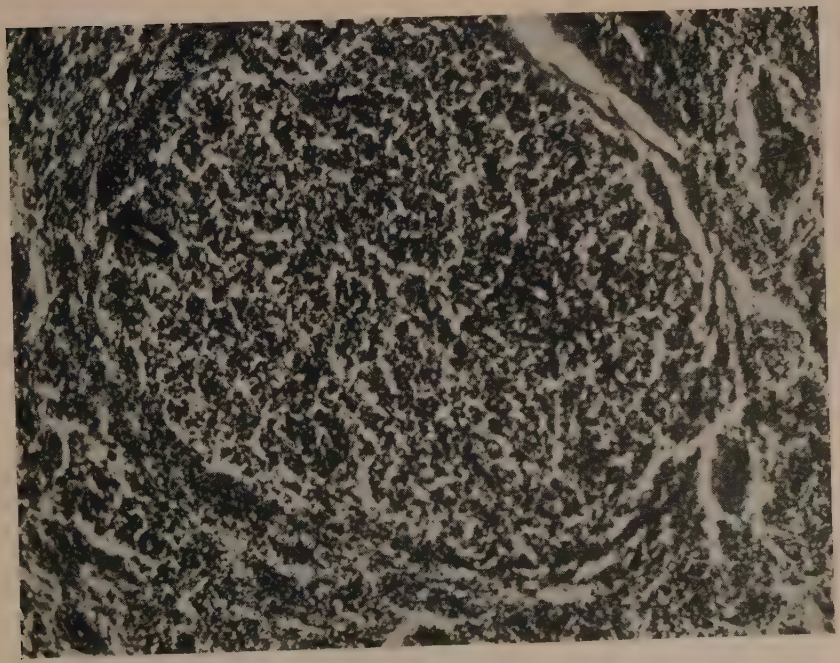


FIG. 2
Section of spleen, showing lymphoid infiltration.

PLATE VII

FIG. 1
Section of kidney, showing lymphoid infiltration.

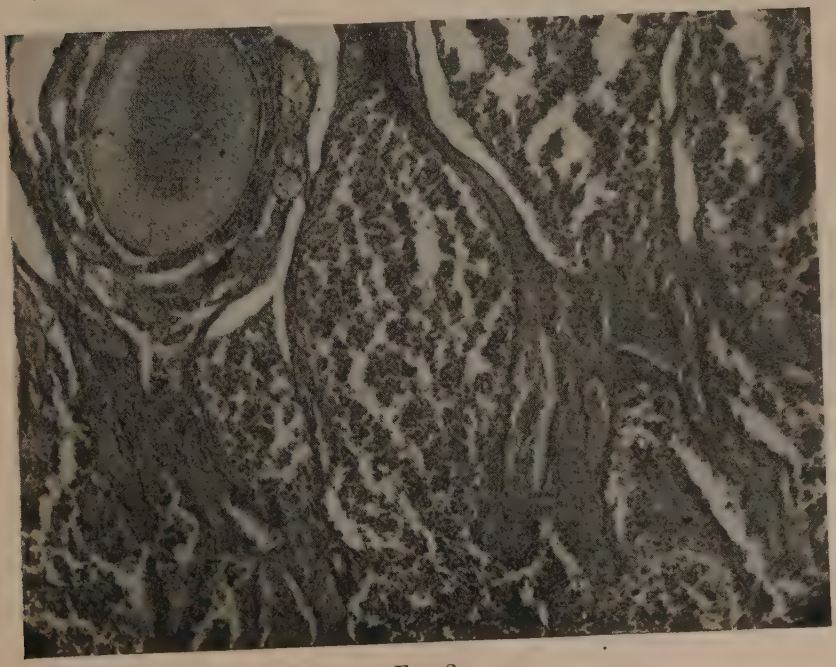


FIG. 2
Section of ovary, showing lymphoid infiltration.

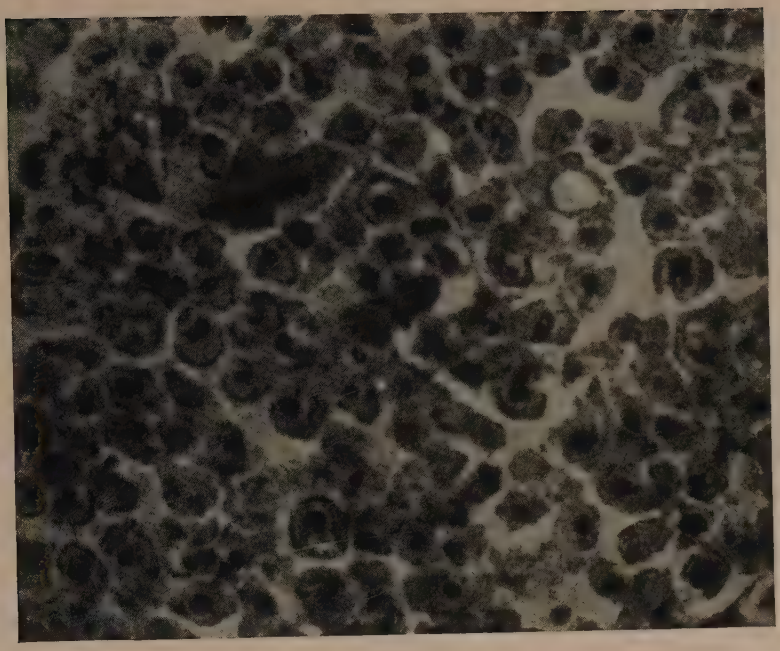


FIG. 1
Lymphoid cells of liver highly magnified.

FIG. 2
Contents of blood vessel highly magnified.

Fig. 2. Ovary from Case No. 4104. Large lymphocytic pseudo leukemia (x 200). In this organ the lymphoid cells have infiltrated nearly everything but the dense connective tissue stroma.

Plate No. VIII. Lymphoid cells from area in liver and from blood vessel.

Fig. 1. Small lymphocytes from Case No. 3448. Pseudo leukemia. (x 1100).

Fig. 2. Contents of a blood vessel in liver from Case No. 2913. Small lymphocytic leukemia (x 900). The lymphocytes appear to be nearly as numerous as the red cells.

ROUP AND CHICKEN POX*

E. M. PICKENS

New York State Veterinary College, Ithaca, N. Y.

Both roup and chicken pox have several synonyms. The more common ones of roup are diphtheria, avian diphtheria, pip, canker, swelled head and distemper. Those of chicken pox are pox, contagious epithelioma and sore head.

A standard text book on medicine, in discussing these diseases, makes the following preliminary statements: "Until very recently avian diphtheria was considered to be a contagious, epizoötic disease of domesticated fowls, characterized by croupous and diphtheritic pseudo-membranes on the mucous membranes of the head, while chicken pox was held to be a contagious, epizoötic disease in which hyperplastic, epithelial nodules of the skin occurred, especially on the comb and wattles, but in addition to which croupous diphtheritic deposits frequently developed on the mucous membranes of the head. The two diseases were considered as independent. More recent research, however, indicates that they are produced by the same organisms."

This statement is not, as yet, universally accepted, therefore the diseases have been considered individually in this article whenever possible.

History. The history of these diseases is somewhat obscure. It is evident from the literature, that fowls have always been subject to various affections of the head, but the first investigation of this class of maladies seems to have been made by Loeffler in 1884. Since that time roup has been studied by Klimmer, Babes and Puscarin, Eberlein, Lohr and Ducloux. Others have studied diseases known as diptheria in pigeons, fowls and other birds. Roup was investigated by Moore at the Bureau of Animal Industry in 1893–4. It has more recently been studied by many workers, some of whom are as follows: Ward of California, Mack of New York and later Mack and Records of Nevada, Harrison and Strait of Ontario, Bromley and Snook of Ohio, etc.

*Read at the Ninth Annual Farmers' Week, New York State College of Agriculture, Ithaca, N. Y.

Chicken pox has been known for a long time. It was thought in early times that it was related to small pox. Spinola failed to produce it in fowls with cow pox. In 1873 Bollinger gave a very full account of his investigations into its nature. Some of the more recent workers are Carnwath, Schmidt, Uhlenhuth and Manteufel, Mack and Records, etc.

Economic Importance. The economic importance of these diseases is very great, as they are probably two of the greatest hindrances in the poultry business. The direct losses from the diseases vary greatly in different epidemics. Thus, in a virulent outbreak, there may be many deaths in a short time; while in another, a flock may become infected and only a few birds die. Of much greater importance are the indirect losses; and this applies to chicken pox especially. These losses are apt to be overlooked by farmers, or those who keep only a few fowls and pay but little attention to them. The diseased birds recover very slowly; and they remain thin, anaemic and unfit for egg production, fattening or breeding; eating just as much as if they were normal and living at the expense of their keeper.

Fowls Affected. Given in the order of susceptibility, the fowls affected with the two diseases would probably be: pigeons, chickens, turkeys, pheasants, peacocks, geese and water fowl to some degree. The susceptibility is generally greater in young than in older birds. An attack of chicken pox confers protection against subsequent infection, for chickens which have recovered from the artificially produced disease, cannot be reinfected after two weeks. The immunity acquired is, sometimes, only of short duration.

Geographical Distribution. Both diseases are widespread; but they are more prevalent in some places than in others; thus for example, they are said to be almost unknown in the Canadian maritime provinces and in eastern Ontario, while in southern Ontario they are the most prevalent diseases of fowls. In southern Europe (Italy) roup rages with great virulence and sometimes presents a constitutional character while in the northern regions the losses, especially on pedigreed fowl farms, are heavy on account of the frequent severe invasions of the disease and high death rate. In the United States both diseases have been reported in almost

every locality. Sedgwick reports that chicken pox is one of the greatest obstacles to chicken raising in Hawaii.

Etiology. As before stated, some investigators claim that the same germ is the cause of both diseases: namely, an ultra-microscopic virus. This is an organism that will pass through the pores of infusorial filters and cannot be seen with the microscope or grown in visible quantities upon artificial culture media. Carnwath succeeded in producing a diphtheritic disease of the mucous membranes with pure contagious epithelioma material, and with diphtheritic material produced contagious epithelioma. These experimental results were confirmed by Schmid, by Uhlenhuth and Manteufel and by Ratz. Hutyra and Marek consider the two conditions etiologically identical. Bromley and Snook state that the two affections are due to a common cause, viz., the filterable virus. They believe, however, that the secondary invaders are responsible for the serious lesions. They also state that all the old names for these diseases should be discontinued and that one name be used. For this they propose, "Epitheliosis Infectiosa Avium."

There are, however, many investigators who are certain that their results show that the germs causing these diseases are not the same, and that the infection one time will not produce roup and at another chicken pox. Bordet and Fally believe that avian diphtheria has nothing in common with contagious epithelioma and Jowett says that this idea, needless to say, is quite erroneous. Haring and Kofoid conclude that there is good evidence to believe that nasal roup and contagious epithelioma are two distinct diseases; that immunity to one does not confer immunity to the other; that diphtheritic lesions in the mouth and throat of fowls may be produced by either roup, contagious epithelioma or mechanical injury, followed by a mixed infection with various organisms; that fowls may be affected by both diseases at the same time. They do not appear to regard avian diphtheria as a distinct pathological entity, but consider that it may be due to either infection. Several bacteria, as well as protozoa, have also been described as being the causes of the diseases.

Pathogenicity. When finely pulverized pox scabs are rubbed into the skin of the comb, wattles or eyelids of chickens, there occur after five or six days swelling and paleness of the affected

parts and on the following day a swelling as large as a millet seed appears on the surface. After three or four more days hemorrhages suddenly appear over the affected area, later the blood dries, forming thick scabs, beneath which the papilla (the part of the skin principally affected by this disease) enlarge considerably and become covered with a dirty gray exudate. Beneath this bloody scab healing takes place after a shorter or longer period of time; but when the diseased portions are irritated (rubbed or scratched) there develop extensive changes of the papillary tissue which delay healing. The result from inoculation of the filtered virus is similar, only the incubation period is from eight to ten days on account of the dilution of the infectious substance.

When roup material (diphtheritic membranes) is employed for inoculation on the mucous membranes of healthy birds only negative results are generally obtained. Lowenthal and Burnet claim to have produced chicken pox on the skin by injecting pox material into the blood. Uhlenhuth and Manteufel used intravenous injections of pox and roup material and produced severe diphtheritic symptoms on the mucosa of the mouth and in the eye.

Aside from their presence in the pox scabs and the mucous membranes, the viruses are also found in the blood at the beginning of the diseases, for it has repeatedly been found possible to produce the characteristic changes in the skin and mucous membranes, especially with liver substance.

Natural Infection. Both diseases are usually introduced into a flock by exposure of the birds to sick ones at shows or by bringing affected fowls on the premises. The contagion may be carried by birds which have the diseases in so mild a form that they show no symptoms of them. There is a general belief that roup may develop by exposure of birds to draughts of air or by keeping them in damp, filthy and badly ventilated houses. It is presumable that this belief in its cause is not well founded because of confusion existing concerning the early symptoms of the disease and those of simple colds and catarrhs. Dampness and lack of ventilation favor the maintenance of the virus when introduced.

It is possible that chicken pox infection may occur through the unimpaired skin, which is favored, no doubt, by a preceding injury, and the frequent disease of the eyelids may be associated

with the habit chickens have of rubbing the vicinity of the eye with their feet. A healthy fowl may contract the disease from a sick one by contaminated drinking water, food, etc., serving as media of infection.

Post Mortem Findings. In roup the toxin from the areas of the disease is very destructive, as the rapid loss of flesh of the bird following a severe attack shows. Upon examination of the membranes that have formed in the mouth, it will be found that when they are removed there is left a raw, granular appearing surface. Upon microscopical examination, cellular infiltration is seen, with a destruction of cells of the mucous membrane underlying the diphtheritic patch. An examination of the maxillary sinus will reveal it to be filled with pus which is often cheesy-like in consistency. The wall over this part is very thin and can be easily opened with a knife.

A microscopical study of sections of the head through the inflamed area shows considerable thickening and an acute inflammation; at times the entire passage is plugged with mucus.

On examination of the eye there may be seen a cloudy condition of the cornea. There is also an acute inflammation of the conjunctiva. In certain cases it has been found that the acute inflammation spreads to other parts of the eye and its surrounding structures.

In chicken pox, the nodules, which are the visible manifestations of the disease, consist of epithelial cells. There is a cell infiltration of the skin and subcutaneous tissue. The epithelial cells are much larger than normal; they may be in scales or in more dense masses. Later in the course of the disease the nodules may drop off as scales.

Symptoms. Roup generally begins without any marked disturbance of the general condition, with a local affection of one of the mucous membranes of the head, usually the mouth. In some cases these develop on the otherwise smooth surface, which is at times only slightly reddened, small spherical or oval yellowish-white spots, which generally spread and finally form extensive membranes. In other cases the mucous membranes assume a dark red color, soon followed by a gray deposit on its surface which gradually becomes thicker and forms dense masses, at first gray or

yellowish, later brownish gray and dark brown, on the parts exposed to the air; when dry, the surfaces become rough and fissured. The membranes are usually adherent to the subjacent tissue. When they are removed, they leave red, uneven, slightly depressed and bleeding surfaces, or the mucosa is covered with cheesy, easily removed masses of exudate, and beneath these the surface is only reddened and finely granulated, but not eroded.

The adjoining tissue which is not covered with membranes is oedematous, but here also the masses often occur in the later stages; these gradually become confluent and finally completely cover the affected cavity. Such abundant deposits may occur within the first or second day, and after their removal, new ones form; sometimes, however, the ulcerative process extends deeper, leading to extensive destruction. If the fowl survives, the membranes are in the course of time loosened and healing takes place.

Such membranes develop most commonly on the mucosa of the mouth, especially on the gums near the longitudinal cleft, on the border and under the tongue, the corners of the mouth and cheeks, as well as on the mucous membranes of the throat. The inflammatory process may extend from the larynx to the trachea and lungs; on the other hand, it may extend from the pharynx to the mucosa of the esophagus and crop. As the inflammation progresses breathing and swallowing become more and more difficult; the bird keeps its neck constantly extended and the beak always open, or opens it every few moments snapping for air, whereby the inrush of air is accompanied by whistling or rattling noises. In the meantime the desire for food, at first unimpaired, grows less and less and swallowing is more difficult or impossible, the bird ceasing to eat altogether.

The affection of the nasal cavity is at first manifested by a thin nasal discharge which soon becomes thick and later a dirty-gray color. It is discharged in considerable quantity on pressure over the top of the nose; otherwise, it dries in the nasal orifices, closing these in part or entirely and occasionally raising the upper nasal wall. In the meantime, the birds breathe heavily, show a desire to sneeze and shake their heads, thus hurling out masses of pus. From the nasal cavity the inflammation extends to the tear canal which, as a result, becomes plugged; it also progresses quite fre-

quently on one or both sides of the cella infraorbitalis which now becomes filled with exudate and bulges under the internal angle of the eye in the form of a growth, which on pressure discharges pus from the nasal orifice on the affected side. The tumor is hot, painful and in the later stages shows on pressure a hard nucleus.

The constantly increasing swelling forces the orbit upwards and backwards, while the corresponding wing of the palate is arched against the mouth cavity and later is entirely destroyed, so that the cavity appears to be surrounded only by mucous membrane or by the dense false membrane. This produces a marked disfigurement of the head, while the movements of the head or beak are greatly interfered with by the tumor. On opening, the growth discharges a yellowish-white, cheesy or caseated mass, or dry and brittle lumps may be removed from its cavity.

When the eyes are involved, the first indications are acute catarrhal symptoms of the conjunctiva with œdematous, swollen, painful lids beneath which there accumulates much muco-purulent exudate filled with air bubbles, sealing the eyes over night. If the eye slit is not opened at regular intervals the discharge dries, forming thick, yellowish-white caseated masses, which cause a protrusion of the lids and force the eye backwards, forming on the surface of the cornea club-shaped masses whose dry, brownish yellow ends project between the compressed lids. The masses are easily removed, but form again rapidly. In other cases false membranes form on the conjunctiva; the inflammation may also extend to the sclera and from there to the cornea, which becomes opaque or ulcerated, and finally panophthalmia results.

Involvement of the intestinal tract occurs only in the later stages of the disease and is manifested by a profuse diarrhœa with frequent discharges of fluid or creamy excreta, sometimes mixed with pus or blood, and causing a rapid weakening of the fowl. The general condition of the patient is at first not disturbed, but later the condition changes markedly. Unable to eat and gasping for air, the birds sit around tired and depressed, with necks drawn out, wings hanging down, feathers ruffled, and do not resist attempts to catch them. The body temperature is raised only in the late stages, while toward the end it falls below normal. The comb and wattles are at first bluish red, later pale and cold to the touch. The discharge emanating from the mouth and nasal orifices soils the

breast and emits an unpleasant odor. The fowls emaciate considerably, until they finally die from exhaustion due to the associated diarrhœa.

In chicken pox the changes develop in the skin and in the majority of cases the skin of the head is first affected. On comb, ears and wattles, in the vicinity of the natural openings, and especially on the parts of the body which are slightly covered or totally devoid of feathers there arises at first a fine, branlike gray deposit, which is soon followed by little nodules, at first reddish gray in color and with a mother of pearl lustre, but which later assume a more grayish yellow color, and are composed of horny or fatty degenerated epithelial cells. By gradual enlargement these develop to pea-sized, yellow or dark-brown nodules whose surfaces are warty, dry and firm, the interior contents being composed of a yellow, fatty paste. Sometimes they are so abundant that they touch and even coalesce with the result that the slightly reddened and easily bleeding skin forms thick, cohesive scabs. The comb and wattles are in consequence much thickened and deformed. The margins of the nasal orifices and, still more, those of the eyelids also become thickened so that eventually the eyes are completely closed. Should the inflammatory process spread to the conjunctival tissue much caseous and purulent secretion accumulates in the conjunctival sac which gradually effects a pressure atrophy of the eyes, or a corneal inflammation develops, which may become complicated with a purulent panophthalmitis.

Nodules similar to those on the head develop in some cases on other portions of the body, especially in the vicinity of the vent, on the under surface of the wings, on the neck, on the rump, particularly on the unfeathered areas. Here they sometimes attain the size of a hazel nut and may even develop into horny or callous growths. Such large growths which have an irregular surface, may, by the exercise of considerable force, be removed or broken off from the skin, which will expose the interior cavity filled with a thick, cheesy mass, while vertical sections show wavy layers running more or less parallel with the skin surface.

As long as the disease is confined to the skin, the general condition usually remains undisturbed; only when it is widely distributed over the surface of the body marked emaciation is likely to occur.

Those cases in which both the skin and mucous membranes of the head are affected although not necessarily at the same time, may be designated as the mixed form. In the majority of cases, the skin of the head is at first affected, and the inflammatory process usually spreads from the corners of the mouth, later to the oral mucosa and from here to the neighboring cavities; the reverse order is much less commonly observed. No matter in what direction the process progresses, the two forms coalesce in such a variety of clinical pictures that in the lethal cases one usually finds the signs of pox and of diphtheria simultaneous, although developed to different degrees. However, cases in which the mucous membranes alone are affected are not rare.

Course and Prognosis. In roup a good number of cases spontaneously become arrested and cure follows in these instances, but as the process is easily carried to the throat and lungs and often involves the intestinal mucosa the death rate is much higher on account of exhaustion or suffocation. The disease usually lasts two or three weeks, sometimes one to two months, and with transitory improvement the time may be even more prolonged. According to Friedberger the mortality may be as high as 50 to 70%, and even higher in young birds, especially those of highly bred varieties. It depends upon the virulence of the outbreak.

In chicken pox, when the disease is confined to the skin, it usually runs a favorable course. The disease ceases to progress after a certain time when the nodules have become dry and have been shed, and spontaneous cure follows after three to five weeks. In this manner even larger nodules may fall off spontaneously; in other cases there occur fresh nodules on other parts of the body, and in these instances the disease runs over several months.

Differential Diagnosis. The differential diagnosis of roup and chicken pox cannot be made until we are possessed of a knowledge of the cause; and if we accept the theory that they are due to the same cause, namely, a filterable virus, the differentiation is not necessary. At present all affections of the head characterized by diphtheritic membranes are accepted as cases of roup. Chicken pox is differentiated by the nodular character of the lesions. Mason's eye worm (Oxyspirura mansoni) of chickens produces

lesions that might be mistaken for roup. The finding of the worm would determine the diagnosis.

Contagious catarrh of the nose is confined exclusively to the nasal cavity, runs a course without any development of the false membranes and is, therefore, not associated with as severe symptoms as roup. In mycosis aspergillina the deposits on the palate and nose are dryer and sometimes of a greenish color; the fungi are easily demonstrated with the aid of a microscope.

Treatment. For roup local treatment consists in freeing the mucous membranes of their deposits. When the membranes are ripe, this is accomplished by catching with the forceps and carefully stripping off the deposit, or removing it with a piece of cotton on a stick or other object. The exposed surface is painted over once or twice a day with 1% silver nitrate solution (if in the mouth apply an after treatment of saline solution), 1% corrosive sublimate solution (after treatment with soda solution), with lactic acid, lemon juice, 2% creolin or lysol solution, tincture of iodine, or the skin is painted with concentrated glycerine.

The secretion which accumulates in the eyes is washed out with warm water and the eye is subsequently treated with 2% boric acid, tannin or creolin. Tumors below the eyes are to be opened as early as expedient and after thorough evacuation of the cavities these should be thoroughly cleansed.

For difficult respiration, inhalation of turpentine and tar vapors may be tried, while against intestinal inflammation tannin or sulphate of iron (1% solution in drinking water), red wine (spoonful doses) or other astringents; in geese, glycerine may be employed, but in most cases treatment is here of little avail.

In chicken pox where the disease is confined to the skin proper treatment will usually affect a cure, and the affection of the mucous membrane may sometimes be arrested in its incipiency. The nodules and nodes on the skin are softened with ointments, oil or soda solution; and removed in the same manner as described for the false membranes of roup.

Local treatment must be continued until the cleansed skin or mucous membrane shows no nodules or deposits. In those instances where proper treatment is not available or is impracticable, or when the birds are of little value, it will prove more practical to

suppress the disease by killing the diseased fowls or even destroying the entire number of fowls on the farm, and instituting thorough disinfection of the coops.

Vaccination for chicken pox with a preparation made from the scabs of this disease is a rather recent method of treatment, which seems to prove highly satisfactory. It is, as yet, in the experimental stage; and seems to be much more efficient as a preventative than a cure. It will be discussed later.

Prevention. In order to prevent these diseases, it is evident that many conditions must be strictly complied with. The character of the food and the general sanitary conditions, including cleanliness, ventilation and temperature of the poultry houses, must be considered. Undoubtedly there is much to be learned in connection with the proper sanitary care of poultry. In addition to the general sanitary methods, the following rules should be observed:

1. Fowls which have an exudate on any of the mucous membranes, nodules on the head, or which have come from flocks in which such diseases exist or have recently existed, should not be placed among healthy birds.

2. If the disease appears in one or more fowls of the flock, they should immediately be separated from the well ones. If possible, the source of the infection should be determined and removed.

3. The quite common practice of allowing fowls from different flocks to run together during the day should be discouraged.

4. Care should be taken to avoid the possibility of bringing the virus of the diseases from affected flocks in the dirt or excrement which naturally adheres to the shoes in walking through an infected chicken yard. The same care is necessary in the interchange of working implements, such as shovels, hoes and the like.

It is evident to any observer that the fact is too often overlooked that fowls, owing to their method of living, are more liable to infection than other farm animals. This is especially true when they are allowed to run at random, picking up their living from the garbage pile and the barn yard, or securing even more unwholesome food. There is little doubt that many so-called outbreaks of

contagious diseases among fowls are simply enzoötics brought about by the various infections to which they have been exposed.

The control of chicken pox by means of vaccination now seems to be a possibility. A great amount of research work is being done and the results on the whole are very gratifying. Hadley and Beach of Wisconsin have reported some of their findings which are most encouraging. Mack and Records of Nevada have recently published a bulletin on "The Control of Contagious Epithelioma in Chickens by Vaccination," in which they state in part as follows: "In the main, we have succeeded in eradicating the disease from the flocks treated and appear to have materially reduced the mortality among the infected birds. Seven flocks, consisting of 2,878 birds, 2,212 of them apparently well at the time of treatment, but in every instance thoroughly exposed, and in 1,666 visibly infected, and many of them seriously and extensively, have been treated. In flocks containing 3,062 birds the treatment was an unqualified success. In two flocks of 816 birds, trouble followed the injections, and it appears as though the treatment was detrimental. On the whole, however, the results have been satisfactory."

The Agricultural Experiment Station of the University of California has done quite a good deal of work on vaccination for this disease. At the present time, they have a vaccine on the market for chicken pox, which may be purchased by any poultryman in the State. Their vaccine is injected under the skin by means of a hypodermic syringe; and is given in 1 c. c. doses. They recommend that two treatments be given with an interval of five to seven days between.

Bromley and Snook state that instead of preparing the vaccine from the pox scabs and false membranes, better results are obtained by making it from the bacteria obtained from these lesions (the secondary invaders). We quote as follows: "The excellent results derived from the use of a vaccine made from the secondary organisms, both in prevention and treatment, are due to controlling the secondary infections which cause the serious complications. If these are controlled, the infection due to the primary virus is mild and soon disappears."

Material has been freely drawn from the following publications:

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DESCRIPTION OF PLATES

PLATE NO. 1

FIG. 1. Fowl showing eye closed. The conjunctiva is covered with exudate.

FIG. 2. Pigeon showing eye closed. The exudate is also present here.

FIG. 3. Fowl showing nodules of chicken pox.

PLATE NO. 2

FIG. 1. The roof of the mouth of fowl showing the position of the exudate. (Natural size.)

FIG. 2. The floor of the mouth, showing the exudate over the tongue. (Natural size.)

FIG. 3. Section of exudate with subjacent tissues, from mouth of fowl showing destruction of the epithelium and cell infiltration of the underlying tissue. (x17.)

FIG. 4. Sections of cornea of fowl after the removal of the mass of sloughed exudate. (x17.)

PLATE I

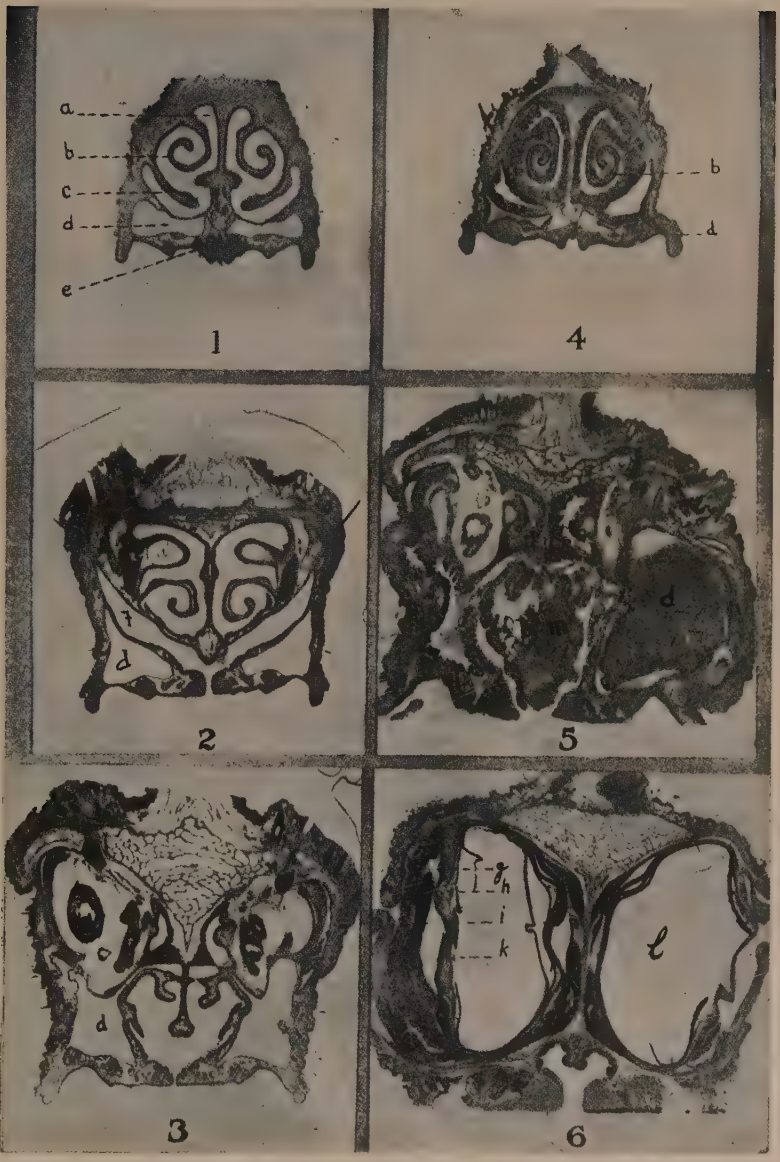
Fig. I

Fig. II.

Fig. III

Lesions of fowls and pigeon.

PLATE III



Sections through the heads of normal and diseased birds.—(Mack.)

Plate No. 3

Photographs of transections of fowls' heads. Figs. 1, 2 and 3 are sections from a normal head. Figs. 4, 5 and 6 are sections from approximately corresponding levels from the heads of fowls suffering with roup.

1. Cross-section of a chicken's head just posterior to the nasal openings. *a*. Nasal passage. *b*. Turbinated bone. *c*. Portion of wall of false nostril. *d*. Sub-orbital sinus. *e*. Palate.

2. Cross-section of a chicken's head midway between the nasal openings and the eyes. *d*. Sub-orbital sinus. d^1. Superior portion of the sub-orbital sinus which connects with *d* posterior to the lachrymal duct. *f*. Lachrymal duct opening into the mouth through the cleft palate.

3. Cross-section of a chicken's head on a level with the anterior part of the eyes. *d*. Sub-orbital sinus and the duct connecting it with the nares.

4. Cross-section of a chicken's head just posterior to the nasal openings, showing the swollen condition of the nasal mucosa in the first stage of the disease. The nasal passage is nearly occluded. *b*. Turbinated bone with swollen mucosa. d^1. Sub-orbital sinus containing a small amount of exudate.

5. Cross-section of a chicken's head midway between the nasal openings and the eyes, showing extensive exudate in the left sub-orbital sinus *d* and nasal passage extending into the cleft palate *m*. The exudate is crowding upon the turbinated bones and nasal septum.

6. Cross-section of a chicken's head through the eyes, showing exudate in the conjunctival sac, inflammatory thickening of the eyelids and membrana nictitans, and ulcerated cornea. *g*. Eyelid. *h*. Membrana nictitans. *i*. Exudate in the conjunctival sac. *k*. Ulcerated cornea. *l*. Eye. (All sections are about twice normal size.)

A STUDY OF FIVE MEMBERS (OR SO-CALLED SPECIES) OF THE SEPTICEMIA HEMORRHAGICA (PASTEURELLA) GROUP OF ORGANISMS WITH SPECIAL REFERENCE TO THEIR ACTION ON THE VARIOUS CARBOHYDRATES.

A. M. BESEMER

From the Department of Comparative Pathology and Bacteriology of the New York State Veterinary College at Cornell University, Ithaca, N. Y.

Many species of animals are susceptible to the disease known as septicemia hemorrhagica or pasteurellosis. This disease has been reported in cattle, horses, reindeer, buffalo, fowls, rabbits and pigs. When an organism has been isolated from an animal infected with septicemia hemorrhagica, it has usually been named according to the animals from which it has been isolated, as *Bact. bovisepticum, Bact. avisepticum,* or *Bact. Renntierpasteurella.*

It is generally considered that the organisms of this group are similar in morphological characters and in many of their biological properties.

The object of this work was to determine whether the members of this group could be differentiated by their cultural properties, especially by their biochemical action on the various carbohydrates.

As this work has been limited to a study of the biochemical characters, the given summary of literature mentions only those works which have taken up these characters in detail.

Table No. 1 gives a summary of the action of the different members of this group on the various carbohydrates, as determined by certain investigators.

Table No. 2 shows the production of indol and phenol as recorded by various bacteriologists.

M'Gowan and Wang have carried out a very extensive study of *Bact. avisepticum* with special reference to the change in the biochemical action of the culture as its virulence was increased by passing through a series of guinea pigs and rabbits. Table No. 3 shows the changes in the cultures after passing through a series of guinea pigs and rabbits. Table No. 4 shows the action of cultures isolated directly from animals dead of septicemia hemor-

TABLE No. 1

SHOWING THE ACTION OF THE SEPTICEMIA HEMORRHAGICA GROUP ON THE VARIOUS CARBOHYDRATES AS REPORTED BY MAGNUSSON, VOURLOUD AND SCHIROP

	Fructose	Galactose	Glucose	Mannose	Sorbose	Arabinose	Xylose	Rhamnose	Lactose	Maltose	Sucrose	Raffinose	Amylose	Dextrin	Inulin	Glycerine	Erythrit	Adonite	Dulcit	Mannit	Sorbit	Amygdalin
1. Magnusson																						
Renntierpasteurella	*	*	*	*	—	—	—	—	*	—	*	—	—	—	—	—	—	—	—	*	*	
Bact. bovisepticum	*	*	*	*	—	—	—	—	*	—	*	—	—	—	—	—	—	—	—	*	*	
Bact. avisepticum	*	*	*	*	—	—	—	—	*	—	*	—	—	—	—	—	—	—	—	*	*	
2. Vourloud																						
Bact. avisepticum	*	—	*			—	—	—	—	—	*		—	—	—	—	—	—	—	*	*	*
3. Schirop																						
Schweineseuche	—	tr.	*			—				—	*	—				—	—			*	*	
Geflügecholera	*	tr.	*			—				—	*	—				—	—			*	*	

* Acid production. — No acid production. tr. Trace of acid.

TABLE No. 2

GIVING THE VARIATIONS IN THE PRODUCTION OF INDOL AND PHENOL AS REPORTED BY A FEW AUTHORS

Author	Organism	Indol	Phenol
Flügge	Bact. suisepticum	—	—
	Bact. buffaloseuche	*	—
	Bact. renntierpasteurella	*	*
Buchanan	Bact. avisepticum	*	*
	Bact. suisepticum	*	*
	Bact. bovisepticum	*	*
Lignières	(Invariable characters)	—	—
Magnusson	Bact. renntierpasteurella	—	
	Bact. avisepticum	*	
	Bact. avisepticum	*	
Moore	Bact. cuniculicida	*	*
	Bact. suisepticum	*	
	Bact. bovisepticum	*	
Smith	Bacteria of swine plague	*	*

*Present. —Absent.

TABLE No. 3

SHOWING THE CHANGES IN BICHEMICAL CHARACTERS OF BACT. AVISEPTICUM AFTER PASSAGE THROUGH A SERIES OF GUINEA PIGS AND RABBITS AS REPORTED BY MCGOWAN AND WANG

	Original	After passage through seven guinea pigs	After passage through seven guinea pigs and three rabbits
Lactose	Slight acid	Acid gas	Acid gas.
Glucose	Slight acid	Acid gas	Acid gas.
Maltose	Slight acid	Acid gas	Acid gas.
Mannite	Acid	Acid gas	Acid gas.
Galactose	Slight acid	Acid gas	Acid gas.
Lactose, neutral red	Acid	Acid gas	Acid gas.
Sucrose	?	Acid gas	Acid gas.
Raffinose	?	Acid gas	Acid gas.
Salicin	No change	Acid ?	Acid gas.
Inulin	No change	No change	No change.
Inosite	No change	No change	Acid.
Sorbite	No change	Acid	Acid gas.
Dulcite	Slight acid	Acid ?	Acid gas.
Adonite	?	?	Acid.
L. milk	?	Acid ?	Acid coagulation.
Pep. water	Indol	Marked indol	Marked indol.
Nitrate	Nitrite	Nitrite	Nitrite.
Motility	Absent	?	Motile.

The culture was not modified from the results reported in the last column after further passage through several other rabbits.

TABLE No. 4

SHOWING THE BIOCHEMICAL CHARACTERS OF CULTURES OF BACT. AVISEPTICUM OBTAINED FROM VARIOUS SOURCES AS REPORTED BY MCGOWAN AND WANG

	I Chicken cholera heart-blood and spleen (pure cultures)	II Chicken cholera trachea Same as Case No. 1	III Strain 1 from Braxy case	IV Strain 2 from Braxy case	V From haemorrhagic septicaemia in cattle
Glucose	A and G	A	A	A and G	A and G
Maltose	A and G	A	A	A and G	A and G
Mannite	A and G	O	A	A and G	A and G
Raffinose	A and G	O	A	A and G	A and G
Galactose	A and G	A	A	A and G	A and G
Dulcite	A and G	O	O	O	A and G
Dextrin	A and G	A	—	A and G	A and G
Sorbite	A and G	A	A	A and G	A and G
Lactose	O	O	O	A and G	A and G
Cane	O	O	A	O	A and G
Salicine	O	O	O	O	A and G
Inulin	O	O	O	O	O
Adonite	O	O	O	O	O
Indol	?	O	O	*	*
Nitrites	*	Trace.	Trace.	*	*
Gelatin	No liquefaction.	No liquefaction.	No liquefaction.	No liquefaction.	No liquefaction.
Potato	Yellowish growth.	O	O	Yellowish growth.	Yellowish growth.
Litmus milk	O	O	O	A	Acid and clot.
Morphology	Bipolar cocco-bacillus.	Bipolar cocco-bacillus.	Bipolar cocco-bacillus.	Bipolar cocco-bacillus.	Bipolar cocco-bacillus.
Motility	Present.	Absent.	Absent.	Present.	Present.

A and G = acid and gas fermentation; A = acid fermentation; ? = doubtful presence; O = no change; * = present in marked quantity; — = test not performed.

rhagica upon the various carbohydrates as reported by M'Gowan and Wang. The results of the investigation here reported can not be directly compared with that of M'Gowan and Wang as no attempt was made to increase the virulence of the cultures.

The methods used in the investigation here reported are as follows: The acid fermentation of the different carbohydrates was determined in media prepared from sugar-free meat infusion bouillon with the reaction adjusted to about +.3, Fuller's scale.

To this sugar-free bouillon 1 per cent of the various carbohydrates was added. An increase in acidity was very marked in some of the carbohydrate bouillons after sterilization. A number of tubes of the same carbohydrate bouillon were inoculated with the same culture and incubated at 37° C. At various intervals, as indicated in the tables, tubes were removed and titrated. Also tubes which were not inoculated were titrated to serve as checks. The results of the titrations are expressed in the number of cubic centimeters of twentieth normal solution* required to neutralize five cubic centimeters of the culture. A minus (—) indicates an alkaline reaction, and no sign indicates an acid reaction in the culture.

The ten cultures used in this work are designated by the names given them by the bacteriologist who isolated the cultures. These had been held as stock cultures for varying lengths of time. The source of these cultures is as follows:

Bact. bovisepticum was isolated August, 1914, from a case of septicemia hemorrhagica in a cow;

Bact. avisepticum, J 1 and J 2, were isolated about 1911 from cases of chicken cholera;

Bact. suisepticum was isolated about 1914 from the lung of a pig;

Bact. Kälberpasteurella A-B and *Bact. Renntierpasteurella* A-B-C, were received from Magnusson about February, 1915.

Bact. septicemia hemorrhagica was isolated about 1910 from a cow.

Table No. 5 gives the results of the study of the action of these cultures upon twenty carbohydrates as determined in this investigation.

* = N/20 NaOH or N/20HCl.

Cultures of *Bact. avisepticum* (*JZ*) and *Bact. Kälberpasteurella* (*Magnusson A*) were inoculated into rabbits. The rabbits died within twenty-four hours, and pure cultures of the organisms were recovered from the heart, liver, spleen and kidney. Subcultures were made in agar plates, litmus milk, lactose bouillon fermentation tubes. No indication of contamination was found in these cultures nor by microscopic examination. The action upon the carbohydrates, of these two cultures after recovery from the rabbit is recorded in Table No. 6.

All ten cultures produced indol and phenol in sugar-free infusion bouillon. These organisms gave a scant growth in 1 per cent peptone solution, but it was insufficient for obtaining a positive test for indol, phenol or ammonia. However, the growth in the peptone solution was sufficient to demonstrate the reduction of nitrates.

Gelatin was not liquefied by any of the cultures within thirty days at 20° C. Gelatin cultures, after being incubated at 37° C. for thirty days, solidified on cooling.

Both litmus milk and plain milk were not apparently changed by any of the cultures. Titrations, however, show that the two lactose fermenting cultures (*Bact. Kälberpasteurella* A and B) produced a small but distinct increase in acidity. The probable reason why apparent changes are not produced in milk is the poor growth in any media having a high acid reaction. Difficulty was encountered in obtaining growth in bouillon when the acidity was seven or eight-tenths (Fuller's scale). The normal reaction of even very fresh milk exceeds this.

At the beginning of this work, each of the ten cultures was inoculated into dextrose, lactose, sucrose and mannite bouillons in Smith fermentation tubes. No gas was produced in any of these. It was assumed from this and from the records of previous investigations that gas was not produced in these carbohydrates. The titrations here recorded were not made from fermentation tubes.

Sorbit could not be obtained. Some of the other chemicals, as dulcit, were so expensive that very few titrations were made with each of these. (See Table No. 5.)

Table No. 7 gives a summary of the action of the cultures upon the various carbohydrates.

In general, the results of this investigation correspond to those of Magnusson. Magnusson, however, reports acid fermentation by *Bact. Renntierpasteurella, Bact. bovisepticum* and *Bact. avisepticum*, while in this investigation *Bast. Kälberpasteurella* only produced acid from lactose. *Bact. Kälberpasteurella* and *Bact. Renntierpesteurella* were received from Magnusson.

Table No. 7 shows acid production from xylose by all cultures studied. This differs from the results of Magnusson and Vourloud. In this study it was found that xylose greatly increased the acidity of bouillon on sterilization, and the cultures would not grow in this acid bouillon. If the initial reaction of the bouillon was such that the final acidity was not too high, the cultures grew and produced acid. From this it is possible that differences in technique may account for different results with xylose.

TABLE No. 5

ACTION OF TEN MEMBERS OF THE SEPTICEMIA HEMORRHAGICA GROUP UPON TWENTY CARBOHYDRATES

1. BACT. SUISEPTICUM (No. 1)

Days incubated	Dextrose		Lactose		Sucrose		Mannit		Dextrin		Inulin		Galactose		Levulose		Glycerine		Raffinose	
Check	.7	.7	.7	.6	.4	.4	.5	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	1.5	1.5	.6	.5	2.1	...	1.8	1.7	.8	.8	.7	.7	1.1	1.0	.9	1.0	...	...	...	...
2	2.1	2.2	.5	.7	2.1	2.2	1.9	1.9	...	...	...	...	...	...	...	...	.3	.3	.4	.3
3	2.2	2.2	.5	.5	2.0	2.1	2.0	2.0	.7	.8	.4	.4	2.2	...	2.3	2.3	...	...	...	...
4	2.2	2.1	.6	.4	2.2	2.2	1.9	1.9	...	...	...	...	...	...	...	...	...	...	...	...
5	...	...	...	...	...	...	...	...	.5	.4	.2	.1	2.2	2.5	2.3	2.2	−.1	−.1	−.1	−.1
7	2.3	2.1	.4	.4	2.1	2.3	2.1	2.0	...	...	...	...	...	...	...	...	...	...	...	...
10	2.6	2.5	.0	−.1	2.4	2.3	2.2	2.2	.1	.3	−.4	−.3	2.2	2.3	2.4	2.4	−.1	−.1	−.3	−.2
30	2.4	2.6	−.1	.1	2.2	2.3	1.9	2.1	−.2	.1	.8	−.4	2.5	2.6	2.2	2.3	1.3	−.1	−.4	.9

2. BACT. BOVISEPTICUM (BAKER)

Check	.7	.7	.7	.6	.4	.4	.5	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	2.3	2.2	.7	.7	2.3	2.2	2.0	2.0	.9	.8	.7	.7	1.7	1.2	2.0	1.9	...	...	...	...
2	2.1	2.1	.8	.9	2.2	2.2	1.9	...	...	...	...	...	...	...	...	...	.3	.3	.4	.3
3	2.2	2.3	.7	.5	2.2	2.2	2.0	2.0	.7	.6	.5	.4	2.7	2.7	2.3	2.7	...	...	...	...
4	2.2	2.1	.6	.7	2.2	2.2	1.9	2.0	...	...	...	...	...	...	...	...	...	...	...	...
5	...	...	...	...	...	...	...	...	.5	.6	.5	.4	2.7	2.8	2.7	2.7	−.1	−.2	−.2	−.2
7	2.4	2.2	.4	.5	2.2	2.3	1.9	2.1	...	...	...	...	...	...	...	...	...	...	...	...
10	2.2	2.5	.2	.3	2.4	2.4	2.3	2.3	.2	.2	−.2	−.2	1.4	2.9	2.7	2.7	−.3	−.3	−.3	−.3
30	2.5	2.4	−.4	.3	2.3	2.4	2.3	2.3	−.1	.6	−.1	.7	2.9	2.5	2.9	2.9	.2	.5	.3	.2

3. BACT. AVISEPTICUM (J 2)

Check	.6	.5	.5	.5	.5	.5	.6	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	1.5	1.7	.6	.5	2.1	2.5	1.6	1.7	.8	.9	.8	.6	1.2	1.5	.9	.9	...	...	...	...
2	2.3	2.3	.6	.6	2.3	2.4	2.1	2.1	...	...	...	...	...	...	...	...	.2	.2	.3	.3
3	2.3	2.2	.5	.5	2.3	2.2	2.2	2.0	.7	.6	.4	.4	2.3	2.3	2.2	2.3	...	...	...	...
4	2.4	2.4	.4	.5	2.3	2.2	2.2	2.1	...	...	...	...	...	...	...	...	...	...	...	...
5	2.4	2.5	.2	.2	2.2	2.3	2.2	2.2	.3	.3	.0	.1	2.3	2.3	2.1	2.1	−.4	−.5	−.5	−.5
7	2.5	2.5	−.3	−.3	2.5	2.6	2.2	2.3	...	...	...	...	...	...	...	...	...	...	...	...
10	2.6	...	−.3	−.3	2.6	2.6	2.2	2.5	.2	.2	−.5	−.5	...	2.6	2.5	2.5	−.5	−.7	−.5	−.5
30	2.2	2.5	.8	.9	2.2	2.3	2.1	2.0	1.0	.4	−.4	.4	2.6	2.4	2.3	2.5	−.4	−.4	−.7	1.1

TABLE No. 5 — (*Continued*)

4. BACT. AVISEPTICUM (J 1)

Days incubated	Dextrose		Lactose		Sucrose		Mannit		Dextrin		Inulin		Galactose		Levulose		Glycerine		Raffinose	
Check	.7	.7	.7	.6	.4	.4	.5	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	2.1	2.1	.7	.5	1.7	2.0	1.7	1.7	.9	.9	.7	.7	1.6*	1.9	.7*	.8	.2	.3	.4	.3
2	2.1	2.1	.5	.5	2.1	2.2	1.9	2.0	...	...	...	...	...	...	...	...	...	...	...	...
3	2.2	2.1	.5	.5	2.2	2.1	2.0	2.0	.7	.6	.5	.4	2.6	2.7	2.2	2.3	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	2.2	2.0	.5	.5	2.2	2.2	2.0	1.9	.5	.5	.5	.3	2.4	2.5	2.5	2.1	−.1	−.1	−.1	−.1
7	2.3	...	.4	.3	2.0	2.1	2.0	2.0	...	...	...	...	...	...	...	...	...	...	...	...
10	2.5	2.4	−.1	−.1	2.2	...	2.3	2.1	.3	.4	−.2	−.4	2.7	2.7	2.5	2.6	−.5	−.6	−.6	−.5
30	2.5	2.3	−.2	−.2	2.0	2.0	2.0	1.9	.4	.4	−.1	.9	2.8	2.8	2.4	2.4	.7	...	1.4	1.1

* Scant growth.

5. BACT. KALBERPASTEURELLA (MAGNUSSON, A)

Check	.6	.5	.5	.5	.5	.5	.6	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	1.7	1.4	2.5	2.2	2.6	2.6	2.0	2.2	...	...	...	...	...	...	...	...	...	...	...	...
2	...	...	2.5	2.5	2.6	2.4	...	...	.7	.6	.4	.4	2.1	2.1	2.1	2.1	.2	.2	.3	.3
3	...	2.4	2.7	2.5	2.3	...	2.2	2.1	.7	.4	.6	.4	2.2	2.1	2.2	2.2	...	...	...	...
4	2.1	2.1	2.5	2.6	2.3	2.3	...	2.3	...	...	...	...	...	...	...	...	...	...	.6	.8
5	2.2	2.4	2.5	2.4	2.3	2.1	2.2	2.1	.3	.4	.4	.2	2.3	2.2	2.2	2.1	−.4	−.5	.3	.8
7	2.5	2.4	...	...	2.5	2.4	...	2.5	...	...	...	...	...	...	...	...	...	...	...	...
10	2.7	2.6	2.7	2.5	2.5	2.6	2.6	2.7	.3	.4	−.3	−.4	2.3	2.3	2.0	2.2	−.6*	−.4	1.0*	1.4
30	2.5	2.4	2.5	2.5	2.6	2.4	2.2	2.3	.2	−.1	.5	...	...	2.4	2.4	...	−.3	−1.3	2.4	2.2

*Sixteen days.

6. BACT. KALBERPASTEURELLA (MAGNUSSEN, B)

Check	.6	.5	.5	.5	.5	.5	.6	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	2.3	2.4	2.3	2.2	2.5	2.5	1.9	2.1	...	...	...	...	...	...	...	...	...	...	...	...
2	...	2.4	2.4	2.6	2.4	2.6	2.2	...	.7	.8	.6	.5	2.1	2.2	2.0	2.1	.4	.3	.4	.2
3	2.3	2.2	...	2.2	2.6	2.6	2.4	2.5	.5	.5	.4	.4	2.4	2.3	2.0	2.1	...	...	...	...
4	2.3	...	2.7	2.6	2.4	...	2.4	2.2	...	...	...	...	...	...	...	...	...	...	...	...
5	...	2.4	2.5	2.6	2.5	...	2.1	2.1	.4	.4	−.1	−.1	2.4	2.3	2.2	2.3	−.4	−.4	.9	.9
7	2.4	2.5	2.6	2.5	2.5	2.4	2.0	2.5	...	...	...	...	...	...	...	...	...	...	...	...
10	2.6	2.6	2.6	2.8	2.4	2.5	2.3	2.2	.2	.2	−.3	−.4	2.5	2.3	2.2	2.3	−.6*	−.4	1.0*	.7
30	2.3	2.5	2.5	2.2	2.5	2.5	2.4	2.3	1.0	2.1	.9	−.3	...	2.8	2.4	...	.9	1.2	2.8	2.9

* Fifteen days.

7. BACT. RENNTIERPASTEURELLA (MAGNUSSON, A)

Check	.6	.5	.5	.5	.5	.5	.6	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.5	.3	.3
1	2.4	2.4	.5	.6	1.6	2.5	2.0	2.0	...	...	...	...	...	...	...	...	...	...	...	...
2	...	2.3	.6	.7	2.4	2.4	2.3	2.2	.8	.8	.4	.6	2.0	2.1	2.1	2.2	.4	.3	.3	.4
3	2.3	2.3	.5	.6	2.2	2.2	2.2	2.3	.8	.7	.4	.4	2.2	2.3	2.0	2.0	...	...	...	...
4	2.2	2.2	.6	.6	2.4	...	2.2	2.1	...	...	...	...	...	...	...	...	...	...	...	...
5	2.4	...	.7	.5	2.4	2.3	2.2	2.2	.4	.3	−.1	−.1	2.3	2.5	2.2	2.2	−.2	−.2	−.2	−.2
7	2.4	...	.7	.5	2.5	2.5	2.1	2.2	...	...	...	...	...	...	...	...	...	...	...	...
10	2.4	2.4	−.3	−.3	2.5	2.5	2.3	2.6	.2	.2	−.4	−.4	2.6	2.4	2.4	2.5	.8*	.8	−.3*	−.2
30	1.0	1.0	−.5	...	2.2	2.6	2.1	2.1	.6	.5	.5	−.4	2.9	...	2.6	2.0	2.3	1.4	2.8	1.4

* Fifteen days.

8. BACT. RENNTIERPASTEURELLA (MAGNUSSON, B)

Check	.8	.9	.9	.8	.6	.5	.5	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	2.6	2.6	.9	.9	2.6	2.4	1.2	...	...	...	...	...	...	...	...	...	...	...	...	...
2	2.3	2.4	.9	.9	2.4	2.6	1.2	2.1	.8	.8	.5	.6	1.2	1.2	2.3	2.2	.3	.3	.3	.4
3	2.4	2.2	.8	.5	2.6	2.5	2.1	2.1	.7	.8	.4	.4	2.2	2.2	2.2	2.3	...	...	...	...
4	2.4	2.3	.3	.4	2.5	2.5	2.1	2.2	...	...	...	...	...	...	...	...	...	...	...	...
5	...	...	...	...	...	...	...	...	.4	.4	−.1	−.3	2.2	2.3	2.2	2.3	−.1	−.2	−.6	−.4
7	2.8	2.8	.1	.2	2.5	2.5	1.9	2.1	...	...	...	...	...	...	...	...	...	...	...	...
10	3.0	2.9	−.1	−.1	2.7	2.7	2.2	2.2	.3	.2	−.4	−.4	2.4	2.5	2.2	2.3	−.1*	−.3	−.4*	−.5
30	3.3	3.1	1.2	1.0	3.0	3.0	1.6	2.6	.4	.2	.9	−.2	2.7	2.8	2.6	2.8	1.5	2.0	2.5	1.8

* Fifteen days.

9. BACT. RENNTIERPASTEURELLA (MAGNUSSON, C)

Check	.8	.9	.9	.8	.6	.6	.5	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	1.6	2.1	.9	.9	2.6	2.5	1.6	1.3	.8	.7	.8	.7	1.2	1.2	2.2	2.2	...	...	...	...
2	2.5	2.4	.7	.7	2.6	2.6	2.0	2.0	...	...	...	...	...	...	...	...	.4	.4	.4	.4
3	2.6	2.5	.7	.3	2.6	2.6	1.9	2.0	.7	.5	.5	.5	2.4	2.4	2.1	1.9	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	2.5	2.5	.4	.2	2.6	2.5	2.0	1.9	.5	.4	.6	.5	2.2	1.8	2.4	2.4	−.1	−.1	−.1	−.2
7	2.8	2.7	−.1	−.2	2.7	2.7	2.1	2.2	...	...	...	...	...	...	...	...	...	...	...	...
10	2.7	3.0	−.3	−.3	2.8	2.8	2.3	2.0	.3	.4	−.6	−.5	2.5	2.6	2.4	2.4	1.0*	1.0	−.2*	−.2
30	3.0	2.9	−.2	.7	2.8	2.8	2.2	2.2	1.2	.9	−.3	.8	2.5	2.7	2.3	2.8	1.1	1.2	2.2	...

* Fifteen days.

10. BACT. SEPTICEMIA HEMORRHAGICA (BOVINE)

Check	.8	.9	.9	.8	.6	.6	.5	.5	.7	.8	.7	.7	.9	.9	.8	.8	.4	.3	.3	.3
1	2.4	2.5	.9	.9	2.4	2.6	1.3	1.5	.8	.6	...	...	...	...	...	...	...	...	...	...
2	2.5	2.6	.8	.8	2.6	2.5	2.0	2.0	.6	.6	.4	.5	1.6	2.0	2.3	2.3	.4	.3	.2	.3
3	2.4	2.4	.6	.6	2.4	2.6	1.9	1.9	.6	.6	.4	.4	2.0	2.1	2.0	2.1	...	...	...	...
4	2.5	2.5	.4	.4	2.6	2.7	2.0	1.9	...	...	...	...	2.1	2.3	...	...	...	...	...	...
5	...	...	...	...	...	...	...	...	.4	.2	.1	.2	2.1	2.3	2.1	2.1	−.8	−.6	−.8	−.8
7	2.7	2.8	.3	.3	2.7	2.6	2.1	2.0	...	...	...	...	...	...	...	...	...	...	...	...
10	2.8	2.8	.3	−.2	2.7	2.7	2.2	2.2	−.1	−.1	−.2	−.5	2.2	2.3	2.3	2.4	−.8*	...	−.7*	−.7
30	3.3	3.0	−.3	...	2.8	2.9	2.6	2.2	1.4	.2	.3	...	...	2.3	2.5	2.4	.7	1.0	2.2	...

* Fifteen days.

TABLE No. 5 — (*Continued*)

1. BACT. SUISEPTICUM (No. 1)

Days incubation	Amygdalin		Mannose		Maltose		Arabinose		Salicin		Xylose		Dulcit		Isodulcit		Adonite		Erythrol		Milk	
Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.3	.3	2.6	2.6	.3	.2	.2	.2	−.3	−.3	...	...	−.3	−.2	−.1	−.2	.2	.1	.1	.1	1.1	1.0
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	−.2	−.3	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	.2	.0	2.7	2.8	−.3	−.3	.2	.2	−.3	−.1	1.9	1.6	.1	.1	−.3	−.3	−.2	−.2	−.2	−.2	1.1	1.1
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	−.3	−.3	3.4	3.3	−.6	−.4	...	...	...	...	1.3	1.9	...	...	−.4	−.4	−.2	−.3	−.2	−.3	1.3	1.2
30	.3	.9	2.8	2.8	−.6	−.4	...	...	...	...	.2	.2	...	...	...	...	.6	.7	.3	.3	1.4	1.3

2. BACT. BOVISEPTICUM (BAKER)

Days incubation	Amygdalin		Mannose		Maltose		Arabinose		Salicin		Xylose		Dulcit		Isodulcit		Adonite		Erythrol		Milk	
Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.4	.4	2.6	2.7	.2	.2	.2	.1	−.3	−.3	...	...	−.3	−.2	−.3	−.3	.1	.2	.1	.1	1.1	1.1
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	−.2	−.3	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	−.1	.1	2.9	2.8	−.3	−.3	.2	.2	−.1	−.1	1.9	2.0	.2	.2	−.3	−.3	−.2	−.2	−.3	−.2	1.3	1.2
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	−.1	−.1	3.3	3.3	−.5	−.6	...	...	...	...	2.0	1.4	...	...	−.2	−.2	−.2	−.3	−.2	−.3	1.2	1.2
30	.1	1.0	3.6	3.1	−.4	−.1	...	...	...	...	2.1	2.1	...	...	...	...	.4	.5	−.3	−.3	1.4	1.3

3. BACT. AVISEPTICUM (J 2)

Days incubation	Amygdalin		Mannose		Maltose		Arabinose		Salicin		Xylose		Dulcit		Isodulcit		Adonite		Erythrol		Milk	
Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.4	.5	2.6	2.7	.2	.2	1.9	2.0	−.3	−.3	...	...	2.2	2.3	−.2	−.2	.0	.1	−.2	.0	1.2	1.2
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	−.4	−.4	2.7	2.7	−.4	−.4	2.0	1.8	.2	.2	1.5	1.5	2.0	1.9	−.3	−.5	−.5	−.5	−.5	−.5	1.2	1.2
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	−.3	−.3	2.7	3.4	−.7	−.7	...	...	...	...	1.3	1.4	...	...	−.7	...	−.5	−.6	−.6	−.4	1.2	1.2
30	1.1	.1	3.4	3.1	−.3	−.1	...	...	...	...	1.7	1.5	...	...	...	...	.3	.3	−.2	.3	1.3	1.4

4. BACT. AVISEPTICUM (J 1)

Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	.4	.5	2.6	2.6	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	...	...	...	...	.25	.25	.2	.1	−.3	−.3	...	...	−.3	−.2	...	...	.1	.1	.1	.1	1.1	1.1
3	...	...	...	...	...	...	...	...	...	...	...	...	−.3	−.3	...	...	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	.2	.3	2.5	2.7	.2	.2	.3	.3	−.1	−.1	1.7	1.7	−.2	−.5	−.3	−.3	−.3	−.2	−.2	−.2	1.0	1.0
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	.3	.3	3.5	3.4	−.3	−.4	...	...	...	...	1.2	1.1	...	...	−.2	...	−.3	−.3	−.2	−.4	1.1	1.0
30	1.1	−.1	3.2	3.1	−.1	−.1	...	...	...	...	1.8	1.7	...	...	...	...	.5	.5	.5	−.1	1.3	1.3

5. BACT. KALBERPASTEURELLA (MAGNUSSON, A)

Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.5	.4	2.7	2.6	.2	.3	.1	.2	−.3	−.3	...	...	−.3	−.3	−.1	−.1	.2	.1	.2	.1	1.1	1.1
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	−.1	−.1	2.7	2.7	−.2	−.2	.3	.2	−.1	−.1	1.3	1.0	.2	.2	−.6	−.5	−.4	−.4	−.2	−.2	1.3	1.2
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	−.3*	−.2	3.2*	3.4	−.6	−.7	...	...	...	...	1.2	...	...	...	−.7	...	−.3	−.3	−.5	−.4	1.6	1.6
30	−.3	−.3	3.4	3.2	−.3	−.2	...	...	...	...	1.7	1.6	...	...	...	...	−.2	−.1	−.1	.5	1.8	1.8

* Sixteen days.

6. BACT. KALBERPASTEURELLA (MAGNUSSON, B)

Check	.4	.4	.7	.7	−.3	−.3	2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.4	.5	2.8	...	.3	.3	.1	.2	−.3	−.3	...	...	−.3	−.3	−.2	−.2	.2	.1	.1	.1	1.2	1.0
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	−.3	−.3	2.8	2.8	.3	−.3	.1	.2	.1	.1	1.5	1.2	.3	.4	−.4	−.6	−.2	−.3	−.1	−.2	1.2	1.3
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	−.2*	−.2	3.3*	3.2	−.3	−.5	...	...	...	...	1.6	1.4	...	...	−.3	−.5	−.4	−.4	−.5	−.5	1.6	1.5
30	1.2	.6	3.2	2.9	−.2	−.4	...	...	...	...	1.6	1.7	...	...	...	...	.5	.4	−.2	.4	1.7	1.8

* Fifteen days.

7. RENNTIERPASTEURELLA (MAGNUSSON, A)

Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.4	.5	2.1	...	.3	.4	.1	.2	−.3	−.3	...	...	.1	−.1	−.2	−.2	.3	.2	.1	.1	1.1	1.2
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	−.1	−.1	2.5	2.4	−.2	−.4	.2	.2	.1	.1	1.5	1.3	−.5	−.5	−.2	−.4	−.1	−.1	−.4	−.3	1.1	1.2
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	−.1*	.2	3.2*	3.1	−.3	−.3	...	...	...	...	1.2	1.2	...	...	−.2	−.2	−.3	−.4	−.4	−.4	1.3	1.2
30	.9	.2	1.8	3.2	−.3	−.4	...	...	...	...	1.7	1.6	...	...	...	...	.5	.5	.2	.3	1.3	1.4

* Fifteen days.

TABLE No. 5 — (*Concluded*)

8. RENNTIERPASTEURELLA (MAGNUSSON, B)

Days incubated	Amygdalin		Mannose		Maltose		Arabinose		Salicin		Xylose		Dulcit		Isodulcit		Adonite		Erythrol		Milk	
Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.5	.5	2.6	2.1	.4	.4	.1	.1	−.3	−.3	...	...	−.2	−.3	−.2	−.2	.2	.2	.1	.1	1.1	1.2
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
4	.4	−.5	2.6	2.6	−.4	−.4	.3	.3	.1	.1	...	...	−.5	−.4	−.4	−.5	−.2	−.3	−.4	−.3	1.1	1.2
5	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	−.3*	−.4	3.2*	3.4	−.4	−.5	...	...	...	...	1.4	1.2	...	...	−.3	−.4	−.5	−.4	−.4	−.4	1.1	1.2
30	−.1	−.1	3.1	3.1	−.4	−.2	...	...	...	...	1.8	1.8	...	...	...	...	−.3	−.2	.3	.2	1.3	1.3

* Fifteen days.

9. BACT. RENNTIERPASTEURELLA (MAGNUSSON, C)

Days incubated	Amygdalin		Mannose		Maltose		Arabinose		Salicin		Xylose		Dulcit		Isodulcit		Adonite		Erythrol		Milk	
Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.5	.4	2.4	...	.2	.3	.2	.1	−.3	−.3	...	...	−.3	−.2	−.2	−.2	.2	.2	.1	.1	1.1	1.2
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	−.1	−.1	2.8	2.6	−.4	−.2	.4	.3	−.1	−.1	1.3	1.4	−.4	−.1	−.4	−.4	−.3	−.4	−.3	−.2	1.1	1.2
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	.1*	−.1	3.6*	3.3	−.4	−.5	...	...	...	...	1.5	1.6	...	...	−.4	−.5	−.4	−.5	−.4	−.3	1.1	1.2
30	1.0	−.7	3.2	3.0	−.4	−.4	...	...	...	...	1.6	1.6	...	...	...	...	.5	.4	.3	.2	1.3	1.3

* Fifteen days.

10. BACT. SEPTICEMIA HEMORRHAGICA (BOVINE)

Days incubated	Amygdalin		Mannose		Maltose		Arabinose		Salicin		Xylose		Dulcit		Isodulcit		Adonite		Erythrol		Milk	
Check	.4	.4	.7	.7	−.3	−.3	.2	.2	−.3	−.3	.5	.5	−.3	−.3	−.3	−.3	.2	.2	.2	.2	1.0	1.1
1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
2	.5	.4	1.4	1.6	.3	.2	.2	.3	−.3	−.3	...	...	−.3	−.2	−.2	−.2	.2	.2	.2	.1	1.1	1.1
3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
4	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
5	−.4	−.2	2.8	2.7	−.2	−.2	.3	.2	.1	.1	1.6	1.7	−.4	−.4	−.5	−.7	−.2	−.2	−.2	−.2	1.1	1.1
7	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	−.2*	−.1	2.7*	2.8	−.7	−.7	...	...	...	...	1.3	1.1	...	...	−.6	−.7	−.3	−.3	−.4	−.4	1.2	1.2
30	1.0	.7	3.2	3.0	−.3	−.4	...	...	...	...	1.8	1.9	...	...	...	...	−.3	.2	.4	.5	1.3	1.3

* Fifteen days.

TABLE No. 6

ACTION OF TWO CULTURES AFTER RECOVERY FROM RABBIT UPON THE CARBOHYDRATES

	Dextrose		Lactose		Sucrose		Mannit		Dextrin		Inulin		Galactose		Levulose		Glycerine		Raffinose	
Check	.5	.5	.4	.3	.2	.2	.3	.3	.4	.3	.3	.3	.5	.5	.7	.7	.3	.2	.3	.3
3. Bact. avisepticum (J 2)	2.1	2.2	.3	.3	1.9	2.0	1.8	1.9	.3	.2	-.1	-.1	1.9	1.9	1.4	1.7	-.1	-.1	-.2	.2
5. Bact. kälberpasteurella (A)	2.5	2.4	2.1	2.1	2.1	2.3	1.7	1.8	.2	.2	-.2	-.1	2.0	2.1	2.1	2.2	.3	.3	1.2	1.2

	Amygdalin		Mannose		Maltose		Arabinose		Salicin		Xylose		Dulcit		Isodulcit		Adonite		Erythrol	
Check	.3	.4	.5	.5	.3	.3	.8	.8	.4	.4	.9	.9	.3	.3	.3	.4	.4	.3	.2	.3
3. Bact. avisepticum (J 2)	.1	.1	2.0	1.8	.6	.6	1.9	1.8	-.3	-.3	1.8	1.9	1.8	1.7	.6	.6	-.1	-.1	-.1	-.1
5. Bact. kälberpasteurella (A)	.2	.2	1.7	1.7	.4	.4	1.4	1.6	2.1	2.2	1.7	1.6	.3	.3	1.0	.7	-.3	-.2	-.1	-.1

TABLE No. 7

SUMMARY OF TABLES NOS. 5 AND 6

	Dextrose	Lactose	Sucrose	Mannit	Dextrin	Inulin	Galactose	Levulose	Glycerine	Raffinose	Amygdalin	Mannose	Maltose	Arabinose	Salicin	Xylose	Dulcit	Isodulcit	Adonite	Erythrol
1. Bact. suisepticum (No. 1)	*	–	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–
2. Bact. bovisepticum (Baker)	*	–	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–
3. Bact. avisepticum (J. 2)	*	–	*	*	–	–	*	*	–	†	–	*	–	–	–	*	–	–	–	–
4. Bact. avisepticum (J. 1)	*	*	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–
5. Bact. kälberpasteurella (A)	*	–	*	*	–	–	*	*	–	–	–	*	–	*	–	*	*	–	–	–
6. Bact. kälberpasteurella (B)	*	–	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–
7. Bact. renntierpasteurella (A)	*	–	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–
8. Bact. renntierpasteurella (B)	*	–	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–
9. Bact. renntierpasteurella (C)	*	–	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–
10. Bact. septicemia hemorrhagica (bovine)	*	–	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–
AFTER RECOVERY FROM RABBIT																				
3. Bact. avisepticum (J. 2)	*	*	*	*	–	–	*	*	–	*	–	*	–	*	*	*	*	–	–	–
5. Bact. kälberpasteurella (A)	*	–	*	*	–	–	*	*	–	–	–	*	–	–	–	*	–	–	–	–

* Acid production. † Slow and weak acid production. – No acid production.

The chief differences in the cultures studied are in the fermentation of lactose and raffinose by the two cultures of *Bact. Kälberpasteurella* received from Magnusson and the fermentation of arabinose and dulcit by *Bact. avisepticum* (*J2*). Since these differences exist after recovery from a rabbit, it reduces the possibility of the cultures being contaminated. Also the work was repeated, where differences were found, after the cultures had been plated and recovered from the plates. The second results agreed with the first, and cultural and microscopic examination did not disclose contamination.

The tubes incubated thirty days gave variable results. That is, the duplicates sometimes showed extremely different degrees of acidity or alkalinity. In some instances, the production of acid was marked after thirty days and not at fifteen days. Since the plugs of the tubes incubated thirty days were paraffined while the others were not, this may have modified the action of the organisms, due to conditions respecting the interchange of air. Except in the case of the action of the cultures of *Bact. Kälberpasteurella* or raffinose, nearly the maximum acidity was produced within two days. For these reasons, the titrations made after thirty days' incubation were considered unreliable, and, therefore, if an increase in acidity was not found within fifteen days, it was recorded in Table No. 7 as not fermenting the carbohydrate used in the test.

The results of this work, together with the findings of previous investigators, show a few variations. These variations could probably be accounted for in differences in technique or in slight variations in the cultures, caused, possibly, by different methods of culturing.

The most striking feature brought out by the study of these organisms is that there is a much greater uniformity between the members of this group in their biochemical properties than has been noted in the study of some other groups of bacteria. There seems to be no cultural nor biochemical basis for designating by different names the five members of this group which were studied.

CONCLUSIONS

1. The members of the septicemia hemorrhagica group studied were practically uniform in their biochemical actions.

2. The passing of the organism through a rabbit did not change its biochemical characters, except to a very slight degree.

The author wishes to acknowledge the assistance of Dr. C. P. Fitch, of the New York State Veterinary College, in suggesting this study and in giving advice in carrying out the investigation. This investigation is a continuation of the work of Dr. Fitch, reported in Report of New York State Veterinary College of 1913–1914.

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A STUDY OF THE FERMENTING PROPERTIES OF BACT. PULLORUM (RETTGER) AND BACT. SANGUINARIUM (MOORE)

S. A. Goldberg

Department of Pathology and Bacteriology

The organisms *Bact. pullorum* and *Bact. sanguinarium* are the causes of two important, closely allied diseases of fowls. Recent work on the relation of these organisms seems to indicate that they are closely allied species if not identical.

This investigation has been attempted in order to determine the action of the organisms on the various carbohydrates. Titrations were made at various intervals of time extending over a period of thirty days.

The following biological characters of these two organisms have been described.

Bact. Pullorum	Bact. Sanguinarium
Morphology. Non-motile rods with slightly rounded ends. 1–3.5 microns by .3–.5 microns. Marked Brownian movement.	Non-motile rods with slightly rounded ends. 1–2 microns long. Marked Brownian movement.
Staining characters. Stain readily with ordinary bacterial stains. Gram negative, uniform stain. No spores.	Stain readily with ordinary bacterial stains. Gram negative, peripheral stain. No spores.
Agar plate. Raised shiny convex greyish white colonies 1–2 mm. in diameter at end of 48 hours.	Raised, shiny, convex, greyish white colonies 5–8 mm. in diameter at end of 48 hours.
Agar slant. Moderate raised dull granular growth.	Abundant, raised, shiny smooth growth.
Gelatin. Finely granular growth along line of stab. Does not spread markedly on the surface. Media not liquefied.	Finely granular growth along line of stab. Does not spread markedly on surface. Media not liquefied.
Potato. Very slight or no growth.	Growth more marked.
Milk. Slight acidity. No coagulation or precipitation of casein.	Gradually increasing alkalinity and finally saponification of the media.
Litmus milk. Slight acidity. No coagulation.	Alkalinity. No saponification.
Dextrose and mannite bouillon. Acid and gas or acid and no gas.	Acid and no gas.
Lactose and saccharose bouillon. Slight alkalinity.	Slight alkalinity.
Maltose bouillon. No change.	Acidified.

Toxin production is identical and differences in *immuniological reactions* have not been found. (Smith and Ten Broeck.)

Material. The strains of *Bact. pullorum* were obtained as follows: No. 1 was isolated in 1913. No. 2 was isolated in 1911. Nos. 3 and 4 were recently isolated from chicks received in this laboratory for diagnosis. No. 5 is an atypical strain isolated in 1911.

The strains of *Bact. sanguinarium* were obtained as follows: No. 1, known as Bact. of Fowl Typhoid III, and No. 2, known as Bact. of Fowl Typhoid IV, were obtained from Dr. Theobald Smith. No. 3 was obtained from Dr. Taylor.

Methods. Beef broth was made sugar free by the action of *B. coli.* To this was added 1 per cent peptone and .5 per cent NaCl. The carbohydrates were added in quantities sufficient to make 1 per cent.

To determine gas production Smith's fermentation tubes were used. For acid production ordinary test tubes were employed.

The titrations were made by $\frac{N}{20}$ solutions of NaOH and HCl respectively, phenolphtalein being used as an indicator.

In the accompanying tables, the figures indicate the number of cubic centimeters of $\frac{N}{20}$ NaOH used to neutralize 5 c.c. of the media. A minus sign preceding the number indicates the number of c.c. of $\frac{N}{20}$ HCl used to neutralize 5 c.c. of the media. The column marked *check* indicates the reaction of the media after two days' incubation previous to inoculation. The tubes were titrated in duplicates in each case.

The gas production was determined in four fermentation tubes of each carbohydrate and the average amount of gas recorded.

The action of the various strains of the organisms were uniform in the different carbohydrates with the exception of amygdalin. In this case *Bact. sanguinarium* strains 1 and 2 were inoculated into a different supply of the media than the other strains.

This latter medium turned to a greenish tinge after sterilization and the amount of acid produced was very small as compared with the other strains of bacteria. In order to check these results, this medium was inoculated with the other strains of the organisms and the results obtained were identical with those of *Bact. sanguinarium* 1 and 2. These latter strains were inoculated into a few tubes of the original medium, with results similar to those of the other strains of organisms on this medium.

Bact. pullorum 5 is an atypical strain which did not produce gas in any of the carbohydrates used. It is in this respect similar to the original Rettger strain. The other strains of *Bact. pullorum* produced gas and marked acidity in dextrose, mannite, galactose, levulose, arabinose and mannose. In these carbohydrates all the strains of *Bact. sanguinarium* studied produced marked acidity and no gas. In isodulcite the first four strains of *Bact. pullorum* produced gas and marked acidity while *Bact. sanguinarium* produced only slight acidity at first, the amount of acidity gradually increasing on prolonged incubation.

In dulcite the strains of *Bact. sanguinarium* produced marked acidity and no gas while the first four strains of *Bact. pullorum* produced slight acidity and gradually turned alkaline on prolonged incubation.

In dextrin results similar to those in dulcite were obtained except that the acidity was not so marked.

In lactose, saccharose, starch, sugar free broth, adonite, salicin, inulin, raffinose and erythrol, all the strains of *Bact. sanguinarium* and the first four strains of *Bact. pullorum* produced slight acidity and gradually became alkaline after prolonged incubation.

In glycerine and xylose there was produced slight acidity at first and increased acidity after prolonged incubation. Xylose showed a marked increase in acidity after sterilization before inoculation, and the medium turned to a brownish color.

Smith and Ten Broeck suggest that these organisms may be a species in the making. They said that they cannot affirm at present whether any strains of *Bact. sanguinarium* produce gas when freshly isolated, or whether certain freshly isolated strains of *Bact. pullorum* do not produce gas.

Taylor described a recent outbreak of fowl typhoid in which the causative organism produced acid and no gas in dextrose and mannite. This freshly isolated strain of *Bact. sanguinarium,* apparently, resembles those strains of *Bact. sanguinarium* that have been kept under artificial cultivation for a considerable length of time.

One year from the date of this work an attempt was made to determine whether there was any change in the gas production

of the various organisms. The work was repeated with dextrose, mannite and galactose, and the results obtained were identical with those of the year previous.

The atypical strain, *Bact. pullorum* 5, differed markedly from the other strains of *Bact. pullorum* studied. It produced acid in milk in twenty-four hours and coagulated milk in twelve days. In this coagulum the whey was not separated from the curd. It did not produce gas in any of the carbohydrates used and it produced more marked acidity than the other strains of *Bact. pullorum.* It is so markedly different from the other strains that it is doubtful whether it should be considered as a strain of *Bact. pullorum.*

Conclusion. The preceding data show that the principal differences in the strains of *Bacterium pullorum* and *Bacterium sanguinarium* studied, lie in the fact that *Bacterium pullorum* produces gas in various carbohydrates while *Bacterium sanguinarium* lacks this power in any of the carbohydrates used. This difference appears to be constant. Judging from the present classification of species of bacteria, this difference in gas production as well as their different actions on milk, maltose, dulcite, dextrin and isodulcite seem to indicate that these two organisms are two distinct species of bacteria.

As this paper was going to press, there appeared an article on the subject by Rettger and Koser. In general, their results correspond with those in this paper. In addition they find that these two organisms differ as regards their reaction to the methyl red test when applied to cultures grown in 1% maltose — bouillon, *Bact. sanguinarium* being methyl red positive and *Bact. pullorum* negative.

The lesser gas production obtained by them may be explained by the fact that beef extract bouillon was used as a basis instead of meat infusion bouillon.

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TABLES SHOWING THE ACTION OF THE STRAINS OF ORGANISMS STUDIED ON TWENTY-ONE DIFFERENT MEDIA

		Average gas in 5 days	Check	2d day	3d day	4th day	5th day	10th day	15th day	20th day	30th day
Dextrose	Bact. Sanguin. 1	No.	1.2, 1.2	4.2, 4.2	3.9, 4.0	3.8, 3.7	3.9, 3.9	4.1, 4.1	4.3, 4.2	3.6, 3.5	3.6, 3.7
	Bact. Sanguin. 2	No.	1.2, 1.2	3.9, 3.8	3.5, 3.4	3.7, 3.9	3.8, 3.7	3.8, 3.7	3.9, 3.9	3.5, 3.5	3.5, 3.2
	Bact. Sanguin. 3	No.	1.0, 0.9	3.9, 4.0	4.1, 4.1	4.2, 4.3	4.1, 4.2	4.7, 4.6	3.2, 3.3	3.6, 3.6	3.6, 3.6
	Bact. Pullorum 1	Bubble	1.0, 0.9	4.1, 4.2	4.2, 4.3	4.3, 4.2	4.3, 4.4	4.8, 4.9	4.6, 4.5	3.7, 4.0	4.6, 4.7
	Bact. Pullorum 2	1.5 cm.	1.0, 0.9	2.8, 2.8	3.1, 3.1	3.4, 3.2	3.5, 3.3	3.8, 3.9	4.2, 4.3	4.5, 4.8	5.0, 4.9
	Bact. Pullorum 3	Bubble	1.0, 0.9	4.7, 4.6	4.7, 4.8	4.6, 4.6	4.5, 4.7	5.4, 5.6	4.6, 4.6	4.2, 4.7	5.2, 4.6
	Bact. Pullorum 4	1.5 cm.	1.0, 0.9	4.7, 4.7	4.7, 4.8	4.4, 4.7	4.8, 4.8	5.0, 5.0	5.4, 5.4	5.0, 4.7	5.3, 4.9
	Bact. Pullorum 5	No.	1.0, 0.9	6.5, 7.0	6.4, 7.2	7.7, 7.8	8.0, 8.0	8.6, 8.5	7.0, 7.3	7.6, 7.4	8.1, 8.6
Lactose	Bact. Sanguin. 1	No.	0.5, 0.5	1.0, 1.1	0.9, 1.0	0.6, 0.8	0.7, 0.8	0.2, 0.2	-0.2, -0.1	0.0, 0.3	0.5, 0.9
	Bact. Sanguin. 2	No.	0.5, 0.5	1.1, 1.0	0.9, 0.9	0.7, 0.8	1.0, 0.9	0.7, 0.7	0.3, 0.4	0.7, 0.8	0.5, 0.6
	Bact. Sanguin. 3	No.	1.0, 0.9	1.4, 1.4	1.4, 1.3	1.2, 1.2	1.2, 1.2	0.2, 0.1	-0.1, -0.2	1.0, 0.8	1.8, 0.8
	Bact. Pullorum 1	No.	1.0, 0.9	1.5, 1.6	1.4, 1.5	1.5, 1.6	1.6, 1.2	1.2, 1.1	0.8, 1.4	1.1, 1.1	1.7, 1.9
	Bact. Pullorum 2	No.	1.0, 0.9	1.3, 1.3	1.5, 1.3	1.4, 1.3	1.4, 1.5	1.0, 1.0	-0.2, 0.2	0.4, 0.3	-0.7, 0.1
	Bact. Pullorum 3	No.	0.6, 0.7	0.9, 0.9	0.9, 1.0	0.8, 0.9	0.8, 1.0	0.9, 0.1	-0.1, 0.1	0.3, 0.2	1.2, 0.2
	Bact. Pullorum 4	No.	0.6, 0.7	0.8, 0.9	0.8, 1.0	0.9, 0.9	0.7, 0.9	0.5, 0.4	0.4, 0.2	-0.1, 0.0	0.5, 0.4
	Bact. Pullorum 5	No.	1.0, 0.9	4.7, 4.6	5.2, 5.4	5.4, 5.3	5.7, 5.1	5.7, 6.2	6.5, 5.9	5.0, 5.0	5.1, 4.6
Saccharose	Bact. Sanguin. 1	No.	0.9, 0.9	1.2, 1.2	1.1, 1.2	1.1, 1.0	1.1, 1.0	0.9, 0.6	-0.1, 0.3	1.0, 1.0	1.1, 1.1
	Bact. Sanguin. 2	No.	0.9, 0.9	1.1, 1.1	1.2, 1.2	1.0, 1.0	1.0, 1.2	1.1, 1.1	0.7, 0.6	0.5, 0.5	1.0, 1.0
	Bact. Sanguin. 3	No.	0.9, 0.8	1.2, 1.2	1.1, 1.0	1.0, 0.9	0.9, 0.9	0.5, 0.6	0.0, 0.2	1.1, 0.9	1.2, 1.2
	Bact. Pullorum 1	No.	0.9, 0.8	1.1, 1.2	1.2, 1.2	1.1, 1.0	1.1, 1.0	0.7, 0.6	0.4, 0.2	1.1, 0.6	0.4, 0.7
	Bact. Pullorum 2	No.	0.9, 0.8	1.1, 1.1	1.2, 1.1	1.1, 1.2	1.1, 1.0	0.5, 0.6	0.2, 0.5	-0.2, 0.3	-0.8, -0.1
	Bact. Pullorum 3	No.	0.6, 0.7	0.8, 0.9	1.0, 0.9	0.9, 0.9	0.8, 0.8	-0.5, 0.6	0.3, 0.3	0.3, 0.0	-0.4, 0.3
	Bact. Pullorum 4	No.	0.6, 0.7	0.9, 0.9	0.9, 0.9	0.5, 0.9	0.7, 0.8	0.5, 0.5	0.2, 0.2	0.3, 0.6	0.3, 0.2
	Bact. Pullorum 5	No.	0.9, 0.8	6.0, 6.2	6.3, 6.2	6.3, 6.3	6.5, 6.5	7.0, 6.9	7.4, 7.4	5.8, 6.0	5.8, 6.4
Mannite	Bact. Sanguin. 1	No.	1.0, 1.0	3.6, 3.5	3.3, 3.3	3.3, 3.4	3.4, 3.2	3.6, 3.7	3.7, 3.6	3.2, 3.2	3.0, 3.3
	Bact. Sanguin. 2	No.	1.0, 1.0	3.2, 3.3	3.2, 3.1	3.2, 3.1	3.3, 3.3	3.5, 3.0	3.5, 3.7	3.0, 3.7	3.1, 3.1
	Bact. Sanguin. 3	No.	1.3, 1.5	3.2, 3.0	3.2, 3.3	3.1, 3.2	3.3, 3.4	3.6, 3.8	3.7, 3.1	3.2, 3.6	3.1, 3.0
	Bact. Pullorum 1	Bubble	1.3, 1.5	3.7, 3.7	3.7, 3.8	3.7, 3.8	4.2, 4.0	4.2, 4.3	4.3, 4.2	4.2, 5.0	4.1, 5.5
	Bact. Pullorum 2	Bubble	1.3, 1.5	3.0, 3.1	3.2, 3.1	3.1, 3.2	3.2, 3.3	3.5, 3.5	3.8, 3.8	4.0, 4.5	4.7, 4.1
	Bact. Pullorum 3	Bubble	0.5, 0.6	4.0, 4.1	4.0, 4.0	4.0, 4.1	4.1, 4.0	4.1, 4.3	4.1, 4.1	4.1, 4.8	4.6, 4.6
	Bact. Pullorum 4	1.8 cm.	0.5, 0.6	4.4, 4.4	4.1, 4.1	4.2, 4.3	4.3, 4.5	4.7, 4.6	4.8, 4.7	4.3, 4.1	4.4, 4.1
	Bact. Pullorum 5	No.	1.3, 1.5	4.0, 3.7	3.9, 4.0	4.3, 4.3	4.3, 4.2	4.5, 4.5	5.0, 4.9	4.4, 4.3	5.3, 5.7

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TABLES SHOWING ACTION OF STRAINS OF ORGANISMS — (*Continued*)

		Average gas in 5 days	Check	2d day	3d day	4th day	5th day	10th day	15th day	20th day	30th day
Amygdalin	Bact. Sanguin. 1	No.	0.5, 0.5	0.9, 0.9	0.8, 0.8	0.6, 0.7	0.6, 0.6	0.5, 0.5	0.2, 0.2	0.7, 0.7	0.7, 1.1
	Bact. Sanguin. 2	No.	0.5, 0.5	0.8, 0.8	0.7, 0.8	0.7, 0.8	0.8, 0.9	0.9, 0.9	0.6, 0.7	0.6, 0.8	0.8, 0.8
	Bact. Sanguin. 3	No.	0.7, 0.7	3.6, 3.6	3.6, 3.4	3.7, 3.6	3.3, 3.4	3.1, 3.1	2.3, 2.9	2.1, 0.5	0.8, 0.8
	Bact. Pullorum 1	No.	0.7, 0.7	3.5, 3.6	3.4, 3.7	3.7, 3.7	3.4, 3.2	3.4, 3.6	4.2, 4.5	3.9, 3.9	3.9, 3.8
	Bact. Pullorum 2	No.	0.7, 0.7	3.4, 3.4	3.3, 3.2	3.0, 3.1	3.1, 3.1	3.2, 3.3	3.4, 3.0	4.0, 3.9	4.1, 4.2
	Bact. Pullorum 3	No.	0.7, 0.7	3.4, 3.5	3.5, 3.5	3.3, 3.2	3.1, 3.0	3.1, 3.2	3.9, 3.5	2.5, 2.0	2.0, 2.0
	Bact. Pullorum 4	No.	0.7, 0.7	3.5, 3.5	3.4, 3.5	3.4, 3.2	3.2, 3.3	3.2, 3.2	3.9, 3.9	2.8, 2.8	3.1, 3.0
	Bact. Pullorum 5	No.	0.7, 0.7	3.0, 2.9	3.1, 3.4	2.8, 3.1	3.1, 3.2	4.1, 4.5	5.3, 5.6	5.3, 5.0	5.5, 5.4
Arabinose	Bact. Sanguin. 1	No.	0.6, 0.5	3.4, 3.2	3.5, 3.7	3.6, 3.6	3.6, 3.5	3.6, 3.6	3.8, 3.7	3.6, 3.8	3.5, 3.5
	Bact. Sanguin. 2	No.	0.6, 0.5	3.3, 3.3	3.5, 3.5	3.5, 3.5	3.4, 3.4	3.3, 3.4	3.5, 3.4	3.6, 3.7	3.4, 3.4
	Bact. Sanguin. 3	No.	1.7, 1.6	3.2, 3.2	3.3, 3.2	3.3, 3.4	3.5, 3.4	3.7, 3.6	3.8, 4.1	4.6, 4.2	5.9, 5.8
	Bact. Pullorum 1	1.8 cm.	1.7, 1.6	3.8, 3.7	4.1, 3.6	3.9, 3.9	4.0, 4.0	4.3, 4.4	4.8, 4.9	5.5, 5.4	6.4, 6.8
	Bact. Pullorum 2	2 cm.	1.7, 1.6	3.8, 3.6	3.8, 3.8	3.8, 3.9	3.8, 3.8	4.4, 4.2	4.3, 4.6	5.4, 5.5	7.6, 7.8
	Bact. Pullorum 3	1.1 cm.	1.7, 1.6	3.5, 3.6	3.8, 3.9	3.7, 3.7	3.8, 3.7	4.3, 4.3	4.6, 4.5	5.1, 5.2	6.0, 6.0
	Bact. Pullorum 4	2.3 cm.	1.7, 1.6	3.6, 2.0	4.0, 4.0	3.9, 2.4	4.0, 2.2	4.3, 4.3	4.7, 4.6	5.1, 5.4	6.2, 4.3
	Bact. Pullorum 5	No.	1.7, 1.6	5.6, 5.7	5.8, 5.9	5.9, 5.7	6.7, 6.2	7.0, 6.7	7.3, 7.2	8.0, 8.3	10.4, 10.8
Adonite	Bact. Sanguin. 1	No.	0.3, 0.3	0.5, 0.4	0.7, 0.6	0.6, 0.5	0.6, 0.5	0.4, 0.5	0.3, 0.3	0.2, 0.2	0.2, 0.2
	Bact. Sanguin. 2	No.	0.3, 0.3	0.6, 0.7	0.6, 0.8	0.4, 0.4	0.5, 0.5	0.3, 0.1	0.4, 0.3	0.0, 0.0	−0.1, −0.1
	Bact. Sanguin. 3	No.	0.7, 0.6	1.0, 1.1	0.9, 0.8	0.9, 0.8	0.9, 0.9	0.5, 0.5	0.0, 0.3	0.1, 0.1	0.6, 0.2
	Bact. Pullorum 1	No.	0.7, 0.6	1.0, 1.0	0.8, 0.9	0.8, 0.9	0.7, 0.8	0.6, 0.4	0.2, 0.1	0.1, 0.3	−0.3, 0.2
	Bact. Pullorum 2	No.	0.7, 0.6	0.7, 0.8	0.8, 0.8	0.6, 0.5	0.4, 0.4	0.4, 0.4	0.0, 0.0	0.1, 0.1	−0.1, −0.2
	Bact. Pullorum 3	No.	0.7, 0.6	1.0, 0.9	0.9, 0.9	0.9, 0.9	0.7, 0.8	0.6, 0.5	0.0, 0.0	0.1, 0.1	0.1, 0.1
	Bact. Pullorum 4	No.	0.7, 0.6	0.8, 0.6	0.8, 0.8	0.6, 0.5	0.5, 0.6	0.5, 0.4	0.2, 0.1	0.2, 0.2	−0.1, 0.0
	Bact. Pullorum 5	No.	0.7, 0.6	1.0, 0.9	0.8, 1.0	0.9, 0.8	0.9, 0.8	1.1, 1.1	1.0, 1.0	1.2, 1.3	1.2, 1.1
Dulcite	Bact. Sanguin. 1	No.	0.5, 0.4	3.1, 3.1	3.0, 3.0	2.8, 2.9	2.8, 2.7	2.9, 3.1	2.8, 3.2	2.7, 2.6	2.8, 2.8
	Bact. Sanguin. 2	No.	0.5, 0.4	3.1, 3.1	3.0, 3.0	2.8, 2.8	2.8, 2.9	2.8, 2.9	3.1, 2.9	2.8, 2.9	2.4, 2.5
	Bact. Sanguin. 3	No.	0.4, 0.5	2.8, 2.9	2.5, 2.6	2.6, 2.5	2.7, 2.5	3.2, 3.2	2.9, 3.0	3.6, 4.0	4.8, 5.0
	Bact. Pullorum 1	No.	0.4, 0.5	0.9, 0.8	0.8, 1.0	0.8, 0.8	0.6, 0.7	0.3, 0.3	0.2, 0.1	0.0, 0.0	−0.3, 0.3
	Bact. Pullorum 2	No.	0.4, 0.5	0.7, 0.8	0.7, 0.7	0.5, 0.5	0.5, 0.4	0.4, 0.2	0.2, 0.3	0.0, 0.1	−0.2, 0.2
	Bact. Pullorum 3	No.	0.4, 0.5	0.9, 0.8	0.8, 0.8	0.8, 0.7	0.9, 0.8	0.5, 0.5	0.2, 0.4	0.2, 0.2	0.5, 0.2
	Bact. Pullorum 4	No.	0.4, 0.5	0.8, 0.8	0.8, 0.9	0.7, 0.7	0.6, 0.5	0.4, 0.5	0.3, 0.2	0.1, 0.1	0.3, 0.3
	Bact. Pullorum 5	No.	0.4, 0.5	0.6, 0.8	0.8, 0.8	0.8, 0.9	0.9, 0.7	1.0, 1.0	1.0, 1.0	1.2, 1.1	1.6, 1.8

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TABLES SHOWING ACTION OF STRAINS OF ORGANISMS — (*Concluded*)

		Average gas in 5 days	Check	2d day	3d day	4th day	5th day	10th day	15th day	20th day	30th day
Glycerin	Bact. Sanguin. 1	No.	1.2, 1.1	1.6, 1.6	1.5, 1.6	1.8, 1.8	1.6, 1.8	2.6, 2.6	2.5, 2.8	3.1, 3.0	2.5, 2.3
	Bact. Sanguin. 2	No.	1.2, 1.1	1.5, 1.5	1.6, 1.6	1.7, 1.7	1.7, 1.8	2.1, 2.3	2.6, 1.9	2.4, 2.6	2.2, 1.8
	Bact. Sanguin. 3	No.	0.6, 0.6	1.1, 0.9	1.3, 1.2	1.1, 1.3	1.3, 1.2	2.3, 2.2	2.5, 2.4	3.1, 3.2	4.0, 4.2
	Bact. Pullorum 1	No.	0.6, 0.6	0.9, 1.0	1.2, 1.2	1.0, 1.2	1.5, 1.8	2.5, 2.5	4.4, 4.6	5.0, 4.9	6.0, 5.8
	Bact. Pullorum 2	No.	0.6, 0.6	1.1, 1.0	1.1, 1.2	1.3, 1.1	1.5, 1.5	2.7, 1.9	3.1, 4.1	3.8, 4.2	4.9, 4.8
	Bact. Pullorum 3	No.	0.6, 0.6	0.9, 0.8	1.0, 0.9	1.1, 1.0	1.0, 1.2	1.6, 1.7	2.2, 1.9	3.1, 3.0	3.6, 3.3
	Bact. Pullorum 4	No.	0.6, 0.6	0.9, 0.9	1.1, 1.2	1.0, 1.2	1.5, 1.4	2.5, 2.0	3.1, 2.5	2.4, 2.6	3.5, 3.6
	Bact. Pullorum 5	No.	0.6, 0.6	0.9, 0.9	1.1, 1.0	1.0, 1.0	1.0, 1.0	1.7, 1.6	2.2, 2.1	3.1, 2.9	5.0, 4.5
Sugarfree broth	Bact. Sanguin. 1	No.	0.4, 0.4	0.7, 0.8	0.7, 0.8	0.7, 0.7	0.4, 0.4	0.2, 0.3	0.1, 0.0	0.5, 0.5	0.1, 0.2
	Bact. Sanguin. 2	No.	0.4, 0.4	0.7, 0.7	0.6, 0.7	0.7, 0.6	0.5, 0.4	0.5, 0.5	-0.3, 0.2	0.5, 0.5	0.1, 0.3
	Bact. Sanguin. 3	No.	0.6, 0.5	0.9, 0.8	1.1, 1.0	0.7, 1.0	0.9, 0.9	0.7, 0.7	0.5, 0.6	0.4, 0.5	0.4, 0.4
	Bact. Pullorum 1	No.	0.6, 0.5	0.8, 0.8	1.1, 1.2	0.8, 0.8	0.8, 0.9	0.6, 0.5	0.1, 0.1	0.4, 0.3	-0.3, -0.4
	Bact. Pullorum 2	No.	0.6, 0.5	0.9, 0.8	0.9, 0.9	0.9, 0.8	0.7, 0.7	0.5, 0.5	0.3, 0.4	0.3, 0.2	-0.3, -0.1
	Bact. Pullorum 3	No.	0.6, 0.5	0.8, 0.7	1.0, 0.8	1.0, 1.0	0.9, 0.8	0.5, 0.4	0.4, 0.3	0.2, 0.3	0.0, 0.0
	Bact. Pullorum 4	No.	0.6, 0.5	0.8, 0.8	1.1, 1.0	0.9, 1.0	0.7, 0.7	0.5, 0.7	0.4, 0.4	0.3, 0.2	0.0, 0.0
	Bact. Pullorum 5	No.	0.6, 0.5	0.7, 0.8	1.1, 1.1	1.0, 0.9	0.8, 0.9	1.1, 1.0	1.0, 1.0	1.2, 1.3	1.4, 1.3
Erythrol	Bact. Sanguin. 1	No.	0.4, 0.5	0.6, 0.7	0.7, 0.8	0.5, 0.7	0.6, 0.4	0.2, 0.3	-0.1, 0.0	0.7, 0.7	0.3, 0.2
	Bact. Sanguin. 2	No.	0.4, 0.5	0.7, 0.7	0.7, 0.7	0.7, 0.7	0.6, 0.5	0.4, 0.3	0.2, 0.8	0.3, 0.8	0.5, 0.7
	Bact. Sanguin. 3	No.	0.7, 0.7	0.9, 0.9	1.0, 0.9	0.8, 0.8	0.6, 0.7	0.5, 0.5	0.2, 0.2	0.2, 0.0	-0.2, 0.3
	Bact. Pullorum 1	No.	0.7, 0.7	1.0, 0.9	1.0, 1.0	0.5, 0.5	0.7, 0.6	0.6, 0.5	0.2, 0.0	-0.1, 0.3	0.0, 0.0
	Bact. Pullorum 2	No.	0.7, 0.7	0.9, 0.8	0.9, 0.9	0.8, 0.8	0.6, 0.7	0.4, 0.3	0.1, 0.1	0.0, 0.0	-0.1, -0.2
	Bact. Pullorum 3	No.	0.7, 0.7	0.9, 0.9	1.0, 0.9	0.9, 0.8	0.8, 0.7	0.6, 0.6	0.2, 0.2	1.0, 0.3	0.3, 0.4
	Bact. Pullorum 4	No.	0.7, 0.7	1.0, 0.9	0.9, 1.0	0.8, 0.8	0.7, 0.7	0.6, 0.7	0.3, 0.3	0.2, 0.1	-0.1, -0.1
	Bact. Pullorum 5	No.	0.7, 0.7	0.9, 0.9	1.0, 1.0	0.8, 0.9	0.8, 0.8	1.0, 1.0	1.0, 1.0	1.2, 1.1	1.4, 1.4

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AMYLOCLASTIC ACTIVITY IN THE DOMESTIC ANIMALS WITH SPECIAL REFERENCE TO THE SALIVA OF THE HORSE.

C. E. HAYDEN

Department of Physiology

Much has been said in regard to the starch splitting power of the salivary secretion in the domestic animals. It seems to be agreed upon that the saliva of the dog and cat do not contain a diastatic enzyme. Most workers agree that there is such an enzyme present in the saliva of the pig. Conflicting statements and opinions on the part of workers in Veterinary Physiology have led the author to investigate the activity of the saliva of the horse. While carrying on this work it seemed desirable to inquire into the amyloclastic power of those secretions and excretions of the horse, oxen, sheep and pig supposed to contain amylases. To that end we have carried out a large number of experiments using salivary extract, blood or serum, bile and urine of these animals.

In a previous report[1] on the dog and cat my data showed diastatic power in the saliva, salivary extract, and blood serum of these two animals. The diastatic power of the blood serum was according to the experiments then carried out greater than in either the saliva or the salivary extract. The conclusions there stated were that no enzymes were produced in the salivary glands beyond the amount produced in the other tissues of the body. These results are borne out by a majority of those who have worked upon the amyloclastic power of the body of these animals.

That there is or is not a definite starch splitting enzyme in the saliva of the domesticated animals it seemed to us could be settled by the means now at our command. The evidence in regard to the presence of an amylase in the saliva that digests starch so far that reducing sugar may be demonstrated is mostly negative. That the saliva may contain an inactive enzyme activated upon its entrance into the stomach or intestines is believed by some. The means by which the enzyme is activated, if such a thing does occur, is undetermined. In our work we digested some samples of saliva, salivary extract, blood, bile and urine, with a 2 per cent solution

of calcium chloride to see if it might act as an activator. The reducing power of these samples was compared with that of samples of the same material digested without calcium chloride. Observations in regard to the amylase content of the blood and urine have been made by other workers using the achromic end point as the index of the amount present. The time in which a small amount of these two fluids will carry a smaller amount of 1 per cent soluble starch solution to the achroodextrine stage varies. The amyloclastic power of the blood and urine of the domestic animals has been found to be very slight when compared with the power in like fluids in man in whom the salivary glands secrete a very active ptyalin. From this evidence there would seem to be a direct relationship between the amyloclastic power of blood and urine and the secretion of the salivary glands. It is significant that in the domestic animals in which the preponderance of evidence seems to show that the saliva has no direct starch splitting power the amyloclastic activities of the other eliminations of the body, both secretory and excretory, are low. It is suggested to my mind that if there was an enzyme in the saliva activated lower down in the digestive tract the amyloclastic power of the blood and urine would be in proportion much higher.

King[2] after carrying on such work as has just been discussed concludes that there are variations in the amylolytic power of urine from different species and that the amylase output in the urine per kilo body weight is greater in those animals which secrete both salivary and pancreatic amylase than in those secreting only the pancreatic amylase.

Gould and Carlson[3] decided that the source of serum amylase is not the pancreas but that it may be due to tissue activity. A high content in the dog does not affect metabolism. Serum diastase in the sheep, horse, ox, and goat is very slight. They feel that a decrease in diastastatic power through pancreatic injury is due to a decrease in internal secretion lessening tissue activity from whence the diastase has its origin.

J. Van de Erve[4] found that ligation of the renal artery or section of the renal nerve did not cause a change in the amount of serum diastase.

Ross and McGuigan[5] showed that ether anaesthesia caused a hyperglycemia but no increase in the diastatic power of the blood.

A diet of meat did cause a greater increase in the diastatic content of the blood than a carbohydrate diet.

Vernon,[6] quoting Goldschmidt, says that aseptic horse saliva gave no evidence of sugar after fourteen days digestion with starch. When the saliva was exposed to the action of bacteria it showed diastatic power as evidenced by the sugar produced after digesting starch with the saliva. Lattimer and Warren, according to the same author, obtained no evidence of diastatic activity from the glands of sheep when they were made up with chloroform water but did obtain activity when the glands were made up with dilute acetic acid. The acetic acid alone did not eliminate bacterial action. It may have been a better extractor than the chloroform water. Vernon also showed that filtered human saliva kept antiseptic for fifteen days doubled in diastatic action. Testing the amyloclastic activity of the pancreas in the domestic animals the author showed that the order was pig, dog, sheep and ox. In this connection let it be remembered that the pig is very generally agreed to secrete an active ptyalin in the saliva. That being true, we might reasonably expect the amylase content of the pancreas of the pig to be greater than that of the other animals mentioned. It has been shown that in those animals in which the saliva has direct digestive action upon starch the amyloclastic power of other fluids of the body is correspondingly greater. The diastatic power of the dog's pancreas is shown to be less than that of the pig, and that of the sheep and ox much less active than either. Rachford[7] concludes that the diastatic action of the pancreas is increased by bile because bile neutralizes the effect of sodium carbonate and has some diastatic power in itself. Accounting for some of the reduction obtained where saliva is used in digestion with starch, Moore & Parker[8] say that some lactose is formed in the salivary gland.

Carlson & Ryan[9] have demonstrated that glucose is normally found in the saliva of the cat in traces, that anaesthesia increases it and that there is more in submaxillary secretion than in the parotid. Stimulation of the sympathetic nerve is said to produce more than stimulation of the chorda tympani. In our own work the saliva of the horse was obtained while the animals were under chloroform anaesthesia and some of the heavy reductions that we obtained in this line of work may have been due in part to glucose

present in the saliva. Such checks as we made, however, show that the reduction after digestion was greater in a number of cases than the combined reduction from saliva and soluble starch when tested alone. These workers say that the glucose comes from the blood. The sugar content of the blood increases under anaesthesia and so one could expect an increase of glucose in the saliva.

Patten & Stiles[10] investigated the influence of neutral salts upon salivary digestion. They concluded that magnesium, calcium, and barium accelerated the digestion more than any of the others used. Their work was with the human saliva.

Various experiments have been carried out with the intent to determine the source of the amylases. Carlson and Luckhardt[11] demonstrated that they were discards of the tissue in general. Stocks[12] states that the amount of amylase in the serum is fairly constant. The amount in the urine varies with digestion. The amount is increased in disease of the pancreas and thus a large quantity is taken to mean an abnormal condition of that organ.

Palmer[13] tested a few samples of saliva from the cow for diastatic power. The flow of saliva was stimulated by the injection of pilocarpin or by washing out the mouth with dilute acetic acid. Using either method a mixed saliva was obtained. Saliva and serum from the same animal were tested as to their power to clear a 1 per cent corn starch solution, the length of time in which the starch would give no reaction when tested with a drop of Lugol's solution, and for reducing sugar by boiling some of the digested material with Fehling's solution. He found the enzyme more concentrated in the blood serum than in the saliva. The author's conclusion is that a diastatic enzyme is present in the saliva but is not specific to it. It is unimportant so far as its digestive function is concerned.

Hawk[14] in his classification of the enzymes speaks of two amylases found in the liver, glycogenase changing glycogen to dextrin and maltose, and maltase changing maltose to glucose. The maltase is also found in the blood serum, saliva, pancreatic and intestinal juices and lymph. Johnston[15] says that the bile of the domestic animals has the power to digest small but appreciable amounts of starch and that in some cases the power is well marked. He suggests that inasmuch as the bile enhances the amylase of the pancreas there may be a body in the bile activating the amylop-

sinogen of the pancreas. He further adds that the intermediate and end products formed as a result of the activity of bile on starch are dextrin, maltose and glucose. The statement is made that there is probably a diastatic enzyme present in bile. Smith[16] quoting Hofmeister says that the bile of the ox, sheep, and horse digests starch while that of the pig and dog does not. That the amylase is specific to the bile is not stated by these observers. The amylase may be found in the bile and still not be a product of direct activity of the liver cells.

METHODS.

The amylases of the saliva, salivary extracts, and other substances tested from the ox, horse, sheep and pig could well have been indicated by the achromic end point. If the saliva contains an active starch splitting enzyme, produced by the salivary glands and helping to any great extent in the digestion of carbohydrates, the digestion should go beyond the achroo-dextrin stage. So we choose to express the digestive action in terms of reducing power as measured by a good and easily used reagent as Benedict's single solution. The achromic end point such as established by Wolgemuth, Roberts, etc., had been used in testing some of the substances included in our tables. Claims that the saliva of the horse, ox, or pig contains an active ptyalin are based upon the production of a reducing sugar which would reduce such reagents as Fehling's or Benedict's. That seems to be the logical conclusion, for it has been shown that an amylase carrying the breaking down of starch to the erythro- or achroo-dextrin stage may be demonstrated in any tissue or tissue fluid of the body. Benedict's solution appealed to us because it is easily used, does not deteriorate, is accurate and much more sensitive than other like solutions. A 1 per cent solution of soluble starch (Merck's according to Lintner) was used throughout the work. If an active enzyme exists in the saliva of the horse, digestion for twenty-four hours with soluble starch ought to produce enough sugar so that it might be measured as to quantity. We obtained samples of saliva from the horse only. The flow of saliva was induced by the injection of one grain (sometimes much less) of pilocarpin. The animals from which these samples were obtained were under chloroform anaesthesia

and as a rule had been under for an hour or more. Salivary glands from the horse were from post-mortem cases. Salivary glands, blood samples, urine and bile from the ox, sheep and pig were obtained in the killing room of the Meat Products division of the Department of Animal Husbandry. The salivary glands were ground in a meat grinder and then thoroughly macerated in a mortar with a 1 per cent acetic acid solution. Chloroform water was added as a preservative. The glandular extracts were allowed to stand for 24–48 hours before using in the work of digestion. According to Vernon[6] the activity of the enzymes present in the extracts does not become apparent until twenty-four hours or longer after being made. Blood was obtained by running it into flasks containing a small amount of potassium oxalate. These samples were in many cases whole blood. Some samples of pure serum were used, mostly from the horse. The digestion was carried on from two to twenty-four hours at a temperature of 38–40° and the reaction was neutral or slightly alkaline. The samples of saliva were from the parotid gland and were collected through a glass tube inserted into Stenson's duct.

The Benedict's solution was standardized using a 1 per cent solution of pure dextrose. To measure the amount of reducing sugar produced in digestion the digested material or a portion of it was added to a boiling solution of Benedict's, 10 c.c. in quantity. Enough 1 per cent dextrose was then run into the beaker from a burette to complete the reduction of the reagent. The complete reduction is marked by a white precipitate with the copper sulphate having been entirely reduced and no blue color left. Benedict's solutions, their composition, and uses, both qualitative and quantitative determinations, are well discussed by Hawk[14]. From most of the blood samples both digested and undigested, the proteins were precipitated by boiling with dilute acetic acid. The filtrate or a small portion of it was tested for reducing sugar.

Amyloclastic activity has been found to be at its best at 40° C. Evans[17] states that the human saliva shows maximum amylase action at 45° C. In the use of Benedict's solution we have been careful to boil for one-half minute after the addition of digested material or 1 per cent dextrose solution to the boiling reagent. We have checked the soluble starch solution at frequent intervals using both digested and undigested samples to see if the starch

in itself exercised any reducing power. We have found at times a slight reduction but not near enough to account for the results we obtained in digesting some samples of saliva from the horse and salivary extract from the pig. Palmer[13] noted that pilocarpine had a very slight reducing power. We feel very certain that its affect upon Benedict's would be very little, if any.

TABLE 1

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount sugar per c.c. saliva
Horse I	Saliva	50 c.c.	50 c.c.	24 hours	0.10 grams
Horse II	Saliva	25 c.c.	50 c.c.	24 hours	0.006 grams
Horse III	Saliva	30 c.c.	50 c.c.	24 hours	0.008 grams
Horse IV	Saliva	50 c.c.	50 c.c.	24 hours	0.019 grams
Horse V	Saliva	50 c.c.	50 c.c.	24 hours	0.014 grams
Horse VI	Saliva	50 c.c.	50 c.c.	24 hours	0.002 grams
Horse VII	Saliva	45 c.c.	50 c.c.	24 hours	0.02 grams
*Horse VI	Saliva	30 c.c.	50 c.c.	24 hours	0.004 grams
Horse VIII	Saliva	50 c.c.	50 c.c.	24 hours	0.019 grams
*Horse VIII	Saliva	50 c.c.	50 c.c.	24 hours	0.008 grams
Horse IX	Saliva	40 c.c.	25 c.c.	24 hours	0.008 grams
Horse X	Saliva	50 c.c.	50 c.c.	24 hours	0.017 grams
*Horse X	Saliva	25 c.c.	50 c.c.	24 hours	0.022 grams
*Horse X	Saliva	50 c.c.	50 c.c.	24 hours	0.016 grams
Horse XI	Saliva	30 c.c.	50 c.c.	24 hours	0.002 grams
Horse XII	Saliva	15 c.c.	50 c.c.	24 hours	0.004 grams
Horse XIII	Saliva	45 c.c.	50 c.c.	24 hours	0.022 grams
*Horse XIII	Saliva	45 c.c.	50 c.c.	24 hours	0.017 grams
Horse XIV	Saliva	20 c.c.	50 c.c.	24 hours	0.013 grams
Horse XV	Saliva	7 c.c.	50 c.c.	24 hours	0.008 grams
Horse XVI	Saliva	30 c.c.	50 c.c.	24 hours	0.004 grams
*Horse XVI	Saliva	30 c.c.	50 c.c.	24 hours	0.000 grams
*Horse XVI	Saliva	30 c.c.	50 c.c.	24 hours	0.001 grams

* Refers to a second sample collected from the same animal in a separate bottle and during the same period of secretion. From horses ten and sixteen a third sample was collected.

TABLE 2

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	$CaCl_2$ used	Amount sugar per c.c. saliva
Horse I	Saliva	50 c.c.	50 c.c.	24 hours	3 c.c.	0.01 grams
Horse II	Saliva	25 c.c.	50 c.c.	24 hours	3 c.c.	0.012 grams
Horse III	Saliva	22 c.c.	50 c.c.	24 hours	3 c.c.	0.008 grams
Horse IV	Saliva	50 c.c.	50 c.c.	24 hours	3 c.c.	0.018 grams
Horse V	Saliva	50 c.c.	50 c.c.	24 hours	3 c.c.	0.005 grams
Horse VI	Saliva	30 c.c.	50 c.c.	24 hours	3 c.c.	0.004 grams
Horse VII	Saliva	.....	.....	.....	.....	.....
Horse VIII	Saliva	50 c.c.	50 c.c.	24 hours	3 c.c.	0.011 grams
*Horse VIII	Saliva	50 c.c.	50 c.c.	24 hours	3 c.c.	0.008 grams
Horse X	Saliva	50 c.c.	50 c.c.	24 hours	3 c.c.	0.017 grams
Horse XIV	Saliva	20 c.c.	50 c.c.	24 hours	3 c.c.	0.014 grams
Horse XVI	Saliva	30 c.c.	50 c.c.	24 hours	3 c.c.	0.002 grams

* A second sample collected in a separate bottle during the same period of secretion. The samples reported in this table after digestion with calcium chloride do not show any great differences in amount of sugar produced when compared with the same samples reported in Table I. Horse II shows the only marked increase. Nos. 6 and 14 show slight increases. There is not enough increase, however, to say that the chloride has any influence in activating any latent ptyalin present in the saliva.

TABLE 3

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount 1% dextrose used	Amount sugar per c.c. extract
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.	0.00024 grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.5	0.00004 grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.4	0.00008 grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.4	0.00008 grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	25 c.c.	25 c.c.	2 hours	2.6	0. grams
Sheep.....	Salivary extract...	20 c.c.	20 c.c.	2 hours	2.2	0.0004 grams
Sheep.....	Salivary extract...	20 c.c.	20 c.c.	24 hours	2.4	0.0002 grams
Sheep.....	Salivary extract...	20 c.c.	20 c.c.	24 hours	2.1	0.00025 grams
Sheep.....	Salivary extract...	20 c.c.	20 c.c.	24 hours	2.	0. grams
Sheep.....	Salivary extract...	20 c.c.	20 c.c.	24 hours	2.	0. grams
Sheep.....	Salivary extract...	10 c.c.	20 c.c.	26 hours	1.8	0.0002 grams
Sheep.....	Salivary extract...	20 c.c.	20 c.c.	26 hours	1.7	0.00035 grams
†Sheep.....	Salivary extract...	16 c.c.	20 c.c.	26 hours	2.	0. grams
†Sheep.....	Salivary extract...	16 c.c.	20 c.c.	26 hours	2.	0. grams
Sheep.....	Salivary extract...	20 c.c.	20 c.c.	24 hours	1.6	0.0002 grams

† Same animal.

TABLE 4

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount 1% dextrose used	Amount sugar per c.c. bile
Sheep.....	Bile.....	5 c.c.	None	2 hours	2.6	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	2 hours	2.6	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	24 hours	2.6	0. grams
Sheep.....	Bile.....	5 c.c.	None	2 hours	2.8	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	2 hours	2.4	0.0004 grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	24 hours	2.6	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	2 hours	2.	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	24 hours	1.8	0.0004 grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	24 hours	2.	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	2 hours	2.	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	24 hours	2.	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	2 hours	2.	0. grams
Sheep.....	Bile.....	5 c.c.	10 c.c.	26 hours	1.4	0.0012 grams
Sheep.....	Bile.....	20 c.c.	20 c.c.	26 hours	2.	0. grams
Sheep.....	Bile.....	20 c.c.	20 c.c.	26 hours	1.6	0.0002 grams
Sheep.....	Bile.....	5 c.c.	20 c.c.	26 hours	1.8	0.0004 grams
Sheep.....	Bile.....	20 c.c.	20 c.c.	26 hours	2.	0. grams
Sheep.....	Bile.....	20 c.c.	20 c.c.	26 hours	1.7	0.00015 grams
Sheep.....	Bile.....	5 c.c.	20 c.c.	24 hours	1.7	0.0006 grams
Sheep.....	Bile.....	3 c.c.	20 c.c.	24 hours	.....	0.0003 grams

TABLE 5

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount sugar per c.c. salivary extract
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.000 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.003 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.001 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.003 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.002 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.003 grams
Pig	Salivary extract...	10 c.c.	50 c.c.	24 hours	0.002 grams
Pig	Salivary extract...	28 c.c.	50 c.c.	24 hours	0.004 grams
Pig	Salivary extract...	10 c.c.	50 c.c.	24 hours	0.022 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.004 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.000 grams
Pig	Salivary extrate...	50 c.c.	50 c.c.	24 hours	0.002 grams
Pig	Salivary extract...	15 c.c.	50 c.c.	24 hours	0.006 grams
Pig	Salivary extract...	10 c.c.	50 c.c.	24 hours	0.003 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.005 grams
Pig	Salivary extract...	30 c.c.	50 c.c.	24 hours	0.003 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.004 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.007 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.003 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.012 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.007 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.003 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.007 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.002 grams
Pig	Salivary extract...	50 c.c.	50 c.c.	24 hours	0.004 grams
Pig	Salivary extract...	20 c.c.	20 c.c.	2 hours	0.000 grams
Pig	Salivary extract...	20 c.c.	20 c.c.	24 hours	0.000 grams
Pig	Salivary extract...	20 c.c.	20 c.c.	24 hours	0.0004 grams
Pig	Salivary extract...	20 c.c.	20 c.c.	24 hours	0.0004 grams
Pig	Salivary extract...	20 c.c.	20 c.c.	24 hours	0.0003 grams
Pig	Salivary extract...	20 c.c.	20 c.c.	24 hours	0.0004 grams
Pig	Salivary extract...	20 c.c.	20 c.c.	2 hours	0.0005 grams

TABLE 6

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount sugar per c.c. bile
Pig	Bile	2 c.c.	20 c.c.	24 hours	0.0000 grams
Pig	Bile	5 c.c.	20 c.c.	24 hours	0.0000 grams
Pig	Bile	10 c.c.	None	None	0.0000 grams
Pig	Bile	10 c.c.	20 c.c.	24 hours	0.0004 grams
Pig	Bile	5 c.c.	20 c.c.	24 hours	0.0006 grams
Pig	Bile	5 c.c.	20 c.c.	2 hours	0.0000 grams
Pig	Bile	5 c.c.	20 c.c.	24 hours	0.0004 grams
Pig	Bile	2 c.c.	20 c.c.	3½ hours	0.001 grams
Pig	Bile	2 c.c.	20 c.c.	24 hours	0.0000 grams
Pig	Bile	5 c.c.	20 c.c.	24 hours	0.0004 grams
Oxen	Bile	10 c.c.	None	None	0.0000 grams
Oxen	Bile	10 c.c.	10 c.c.	2 hours	0.0013 grams
Oxen	Bile	10 c.c.	None	None	0.0000 grams
Oxen	Bile	10 c.c.	10 c.c.	2 hours	0.0005 grams
Oxen	Bile	10 c.c.	None	None	0.0002 grams
Oxen	Bile	10 c.c.	10 c.c.	2 hours	0.0003 grams
Oxen	Bile	10 c.c.	10 c.c.	24 hours	0.0000 grams
Oxen	Bile	5 c.c.	None	None	0.0000 grams
Oxen	Bile	5 c.c.	10 c.c.	2 hours	0.0000 grams
Oxen	Bile	5 c.c.	10 c.c.	24 hours	0.0000 grams
Oxen	Bile	5 c.c.	10 c.c.	2 hours	0.0000 grams
Oxen	Bile	5 c.c.	20 c.c.	26 hours	0.0004 grams
Oxen	Bile	5 c.c.	20 c.c.	24 hours	0.0000 grams
Oxen	Bile	5 c.c.	20 c.c.	3½ hours	0.0000 grams

TABLE 7

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Test used	Results
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Very slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Good
Steer	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Oxen	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Very slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Good
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Positive
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Positive
Oxen	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Negative
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Calf	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Cow	Bile	10 c.c.	10 c.c.	24 hours	Folin's	Slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Pig	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Slight
Oxen	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Oxen	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Slight
Oxen	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Pig	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Slight
Calf	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Calf	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Pig	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Pig	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Pig	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Oxen	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Calf	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Sheep	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Sheep	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Oxen	Bile	10 c.c.	10 c.c.	24 hours	Benedicts'	Negative
Sheep	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Sheep	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Negative
Pig	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Very slight
Pig	Bile	10 c.c.	10 c.c.	24 hours	Benedict's	Very slight

TABLE 8

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount sugar per c.c. serum
Horse	Serum	16 c.c.	50 c.c.	– – –	.004 grams
Horse	Serum	24 c.c.	50 c.c.	24 hours	.009 grams
Horse	Serum	30 c.c.	50 c.c.	24 hours	.013 grams
Horse	Serum	25 c.c.	50 c.c.	– – –	.00 grams
Horse	Serum	50 c.c.	50 c.c.	24 hours	.013 grams
Horse	Serum	28 c.c.	50 c.c.	24 hours	.006 grams
Horse	Serum	50 c.c.	50 c.c.	24 hours	.014 grams
Horse	Serum	50 c.c.	50 c.c.	24 hours	.005 grams
Horse	Serum	50 c.c.	50 c.c.	– – –	.022 grams
Horse	Serum	30 c.c.	50 c.c.	24 hours	.01 grams
Horse	Serum	30 c.c.	50 c.c.	– – –	.011 grams
Horse	Serum	30 c.c.	50 c.c.	– – –	.022 grams
Horse	Serum	30 c.c.	50 c.c.	24 hours	.001 grams
Horse	Serum	30 c.c.	50 c.c.	– – –	.013 grams
Horse	Serum	25 c.c.	50 c.c.	24 hours	.002 grams
Horse	Serum	30 c.c.	50 c.c.	24 hours	.006 grams
Horse	Serum	30 c.c.	50 c.c.	– – –	.002 grams
Horse	Serum	50 c.c.	50 c.c.	– – –	.022 grams
Horse	Serum	45 c.c.	50 c.c.	24 hours	.022 grams
Horse	Serum	30 c.c.	50 c.c.	– – –	.006 grams

– – – Indicates no digestion. Some of the undigested samples in this table give as much as samples having been digested.

TABLE 9

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount sugar per cc.. urine
Sheep	Urine	10 c.c.	– – –	– – –	.0004 grams
Sheep	Urine	10 c.c.	10 c.c.	2 hours	.0006 grams
Sheep	Urine	10 c.c.	10 c.c.	24 hours	.0008 grams
Sheep	Urine	10 c.c.	10 c.c.	2 hours	.0008 grams
Sheep	Urine	10 c.c.	10 c.c.	24 hours	.0004 grams
Sheep	Urine	3 c.c.	10 c.c.	2 hours	.0000 grams
Sheep	Urine	3 c.c.	10 c.c.	24 hours	.0000 grams
Sheep	Urine	10 c.c.	10 c.c.	2 hours	.0000 grams
Sheep	Urine	10 c.c.	– – –	– – –	.0002 grams
Sheep	Urine	10 c.c.	10 c.c.	24 hours	.0004 grams
Sheep	Urine	7 c.c.	– – –	– – –	.0000 grams
Sheep	Urine	10 c.c.	10 c.c.	24 hours	.0001 grams
Sheep	Urine	10 c.c.	10 c.c.	24 hours	.00085 grams
Pig	Urine	10 c.c.	20 c.c.	– – –	.0003 grams
Pig	Urine	10 c.c.	20 c.c.	2 hours	.0005 grams
Pig	Urine	10 c.c.	20 c.c.	24 hours	.0007 grams
Pig	Urine	10 c.c.	20 c.c.	– – –	.0004 grams
Pig	Urine	10 c.c.	20 c.c.	2 hours	.0003 grams
Pig	Urine	10 c.c.	20 c.c.	24 hours	.0008 grams
Pig	Urine	10 c.c.	20 c.c.	– – –	.0004 grams
Pig	Urine	10 c.c.	20 c.c.	3½ hours	.0012 grams
Pig	Urine	10 c.c.	20 c.c.	24 hours	.0011 grams
Pig	Urine	10 c.c.	20 c.c.	– – –	.0002 grams
Pig	Urine	10 c.c.	20 c.c.	24 hours	.0004 grams
Oxen	Urine	10 c.c.	20 c.c.	– – –	.0006 grams
Oxen	Urine	10 c.c.	20 c.c.	24 hours	.0007 grams
Oxen	Urine	10 c.c.	20 c.c.	– – –	.0006 grams
Oxen	Urine	10 c.c.	20 c.c.	2 hours	.001 grams
Oxen	Urine	10 c.c.	20 c.c.	24 hours	.0004 grams
Oxen	Urine	10 c.c.	– – –	– – –	.0000 grams
Oxen	Urine	10 c.c.	20 c.c.	2 hours	.0000 grams
Oxen	Urine	10 c.c.	20 c.c.	24 hours	.0018 grams
Oxen	Urine	10 c.c.	20 c.c.	26 hours	.0002 grams
Oxen	Urine	2 c.c.	– – –	– – –	.0000 grams
Oxen	Urine	10 c.c.	20 c.c.	24 hours	.0003 grams
Oxen	Urine	10 c.c.	– – –	– – –	.0000 grams
Oxen	Urine	10 c.c.	20 c.c.	24 hours	.0008 grams
Oxen	Urine	2 c.c.	– – –	– – –	.0000 grams
Oxen	Urine	5 c.c.	20 c.c.	24 hours	.0008 grams
Oxen	Urine	20 c.c.	20 c.c.	24 hours	.0001 grams

– – – Indicates no digestion. Some of the undigested samples in this table give as much as samples having been digested.

TABLE 10

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount sugar per c.c. salivary extract
Horse	Salivary extract	25 c.c.	10 c.c.	2 hours	.0000 grams
Horse	Salivary extract	10 c.c.	10 c.c.	36 hours	.0013 grams
Horse	Salivary extract	10 c.c.	10 c.c.	3 hours	.0004 grams
Horse	Salivary extract	50 c.c.	20 c.c.	24 hours	.00008 grams
Horse	Salivary extract	20 c.c.	20 c.c.	2 hours	.0007 grams
Horse	Salivary extract	20 c.c.	20 c.c.	24 hours	.0003 grams
Horse	Salivary extract	20 c.c.	20 c.c.	24 hours	.0005 grams
Oxen	Salivary extract	– – –	– – –	– – –	.00012 grams
Oxen	Salivary extract	22 c.c.	20 c.c.	2 hours	.00036 grams
Calf	Salivary extract	25 c.c.	– – –	– – –	.0002 grams
Calf	Salivary extract	25 c.c.	20 c.c.	2 hours	.0002 grams
Oxen	Salivary extract	8 c.c.	– – –	– – –	.0000 grams
Oxen	Salivary extract	10 c.c.	20 c.c.	2 hours	.0003 grams
Oxen	Salivary extract	10 c.c.	– – –	– – –	.0000 grams
Oxen	Salivary extract	10 c.c.	10 c.c.	2 hours	.0008 grams
Oxen	Salivary extract	10 c.c.	– – –	– – –	.0000 grams
Oxen	Salivary extract	10 c.c.	10 c.c.	2 hours	.0004 grams
Oxen	Salivary extract	20 c.c.	– – –	– – –	.0000 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	2 hours	.0000 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	.00005 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	2 hours	.0002 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	.00005 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	2 hours	.0000 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	.0002 grams
Oxen	Salivary extract	30 c.c.	20 c.c.	26 hours	.00013 grams
Oxen	Salivary extract	10 c.c.	10 c.c.	2 hours	.0001 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	.0002 grams
Oxen	Salivary extract	10 c.c.	– – –	– – –	.0001 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	.0004 grams

– – – Indicates no digestion. Some of the undigested samples in this table give as much as samples having been digested.

TABLE 11

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	$CaCl_2$ used	Amount sugar per c.c. substance tested
Pig	Blood	5 c.c.	20 c.c.	24 hours	2 c.c.	0.0014 grams
Pig	Salivary extract	20 c.c.	20 c.c.	24 hours	2 c.c.	0.00025 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	2 c.c.	0.0000 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	2 c.c.	0.0002 grams
Oxen	Urine	20 c.c.	20 c.c.	26 hours	2 c.c.	0.0002 grams
Oxen	Salivary extract	30 c.c.	20 c.c.	26 hours	2 c.c.	0.0001 grams
Oxen	Bile	5 c.c.	20 c.c.	26 hours	2 c.c.	0.0000 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	2 c.c.	0.0001 grams
Oxen	Salivary extract	20 c.c.	20 c.c.	24 hours	2 c.c.	0.00025 grams
Oxen	Blood	20 c.c.	20 c.c.	24 hours	2 c.c.	0.0004 grams
Oxen	Urine	5 c.c.	20 c.c.	24 hours	2 c.c.	0.0002 grams
Oxen	Bile	5 c.c.	20 c.c.	24 hours	2 c.c.	0.00035 grams

TABLE 12

ANIMAL	Substance tested	Amount used	Amount 1% starch used	Time of digestion	Amount sugar per c.c. substance tested
Pig	Blood	2 c.c.	20 c.c.	24 hours	0.000 grams
Pig	Blood	2 c.c.	20 c.c.	2 hours	0.0015 grams
Pig	Blood	20 c.c.	---	---	0.00025 grams
Pig	Blood	5 c.c.	20 c.c.	24 hours	0.0014 grams
Pig	Blood	5 c.c.	20 c.c.	24 hours	0.001 grams
Pig	Blood	5 c.c.	20 c.c.	24 hours	0.001 grams
Oxen	Blood	2 c.c.	---	---	0.0000 grams
Oxen	Blood	2 c.c.	20 c.c.	2 hours	0.0000 grams
Oxen	Blood	2 c.c.	20 c.c.	24 hours	0.0000 grams
Oxen	Blood	5 c.c.	10 c.c.	2 hours	0.0000 grams
Oxen	Blood	5 c.c.	10 c.c.	24 hours	0.0000 grams
Oxen	Blood	20 c.c.	---	---	0.0003 grams
Oxen	Blood	20 c.c.	20 c.c.	20 hours	0.0002 grams
Sheep	Blood	5 c.c.	20 c.c.	26 hours	0.001 grams
Sheep	Blood	5 c.c.	20 c.c.	26 hours	0.0008 grams
Sheep	Blood	10 c.c.	20 c.c.	26 hours	0.0002 grams
Sheep	Blood	2 c.c.	20 c.c.	2 hours	0.0000 grams
Sheep	Blood	3 c.c.	20 c.c.	2 hours	0.0000 grams
Sheep	Blood	10 c.c.	---	---	0.0003 grams
Sheep	Blood	10 c.c.	20 c.c.	24 hours	0.0003 grams
Sheep	Blood	14 c.c.	---	---	0.00014 grams
Sheep	Blood	20 c.c.	---	---	0.0002 grams

--- Indicates no digestion. Some of the undigested samples in this table give as much as samples having been digested.

DISCUSSION

Table 1 is a record of samples of saliva taken from sixteen different horses. Some of these samples after digestion completely reduced 10 c.c. of Benedict's solution. Others required the addition of only a slight amount of 1 per cent dextrose to complete the reduction. From five of the horses we collected two different samples of saliva during the same period of secretion. From the horses sixteen and ten we collected the third sample. Ellenberger[18] states that the parotid and submaxillary saliva of both the horse and ox can convert starch into sugar. In the horse the first saliva is the only secretion to contain the active enzyme and as secretion goes on the power of starch conversion is lost. The same author claims that the secretion from all the salivary glands in the pig has the power to convert starch into sugar. From horses six and five we obtained more sugar per c.c. of saliva from the second sample than we did from the first. Considering horses eight, thirteen and sixteen the reverse holds true. The third sample from horses ten and sixteen showed less sugar after digestion than the initial samples did. We do not have enough data on the second and third secretions to base a definite conclusion but it would seem

that if there were only a slight amount of enzyme present it would be more easily demonstrated in the first secretion. The samples of saliva collected were without exception from the parotid gland through a tube inserted into Stenson's duct. The samples were thus from a single gland and any reduction taking place when heated with the reagent would not be due to the mucous of the submaxillary and sublingual glands. It has been shown that traces of glucose are present in the saliva, probably coming from the blood. It is also shown that there is an increase in the sugar of the blood when an animal is under anaesthesia. The horses from which the saliva was obtained were under anaesthesia and so it would not be unlikely that some evidence of reducing sugar would appear. Samples of saliva undigested have shown some reducing power. The soluble starch which was used has at times shown a little. The combined reducing power of saliva and soluble starch has at no time been near equal to the amount of reduction obtained after digesting saliva and starch together. Neither starch nor saliva having been digested separately has shown any marked reducing power. We do not wish to go on record as saying that every sample of saliva showed evidence of the presence of an active ptyalin but some of them did. The investigation as to the activity of the horse's saliva is being continued with an absolute check on every step of the work and with two added lines of experimentation. The results will be given in a future communication.

Table 2 records eleven samples of horse saliva that were digested with 1 per cent soluble starch in the presence of 3 c.c. of 2 per cent $CaCl_2$. There are three samples, horses two, six and fourteen, which show greater reducing power than the same samples digested without the calcium chloride. Horses two and six show twice the reducing power. Horse fourteen shows a little more. The rest of the samples show equal and in some a less reducing power when digested with calcium chloride. There is no reason from this table for saying that calcium chloride acts as an activator for a latent ptyalin if it exists as such in the saliva of the horse.

Table 3 is a record of twenty-seven samples of salivary extract made from the glands of the sheep. Of the twenty-seven samples twenty-six are from different sheep. Sixteen of them after digestion showed no signs of reducing sugar when boiled with the reagent used. Ten of the samples showed a slight reducing power.

No claim can be made for digestive functions when the decimal part of a gram of sugar produced by 1 c.c. of the substance used goes into four places. The salivary extract of the sheep showed no digestive power.

Twenty samples of bile from the sheep gave very similar results. Only eight of the samples had any reducing power whatever. That was insignificant in each case. The presence of an amylase that produces any amount of reducing sugar is not shown for the bile of the sheep in this line of work.

The digestion of soluble starch with thirty-two samples of salivary extract from the glands of the pig was much more fruitful. The amount of reduction shown in this series is not so great as might be expected in view of the fact that every one seems to be agreed that the saliva of the pig contains an active starch splitting enzyme. Almost all of these samples show signs of digestion. A few glands do not appear to have shown any enzyme. At least twenty-five of the samples did. The glands from which the extracts were made were obtained from pigs slaughtered in the practice work of the Department of Animal Husbandry. The animals had been subjected to the scalding process and the tissue may have been affected somewhat by the heat of the vat.

The samples of bile taken from the pig and oxen and recorded in Table 6 gave about the same amount of reduction as did the bile of the sheep. Bile from any of these animals in our work does not appear to show any richer content of amylase than might be expected from the blood or urine of the same animals Bile should show a marked digestive action according to the opinions expressed by the most observers. The tests that are recorded in Table 7 were made with 36 samples of bile. Folin's[19] test is said to be especially sensitive. Benedict's is also very sensitive. These tests were used because it was thought the color of the bile might hide the point at which the copper was completely reduced in the quantitative test. The bile was digested with soluble starch. Some of this digested solution and the reagent were boiled together for a sufficient length of time to insure any reduction that might take place. A centrifuge tube was filled with the boiled solution and centrifuged for two minutes. The amount of reduction was measured by the amount of oxide settling down in the centrifuge tube. Of the samples tested in this manner only two showed what

were called good results. Most of them were slight, very slight, or negative. The first two mentioned were the only ones to show enough oxide to indicate a small amount of digestion. Samples of bile from oxen, pig, calf and sheep were tested in this way. The results in this series indicate that the amylase content of the bile is not great enough to make the digestion of starch by the bile of very great importance.

The serum of the horse gave varying results. The amount of sugar found in either the digested or undigested samples was not very satisfactory to the author. The amylase content of the blood, however, does not appear to be great enough to produce an appreciable amount of sugar when digested with soluble starch. The same conditions prevail in the whole blood. The record of the whole blood samples are shown in Table 12.

Neither does the amylase content of the urine appear to be great enough to digest starch beyond the achromic end point. Table 9 does not show any great amount of reduction per cc. of urine digested. The samples were taken from the sheep, pig and oxen.

Table 10 records the results obtained after digesting the salivary extract from the horse and oxen. There is not enough reduction on the part of any of these samples to indicate much amylolytic action.

A few samples of various substances were digested with 2 per cent calcium chloride. These samples, excepting the saliva of the horse, are recorded in Table 12. None of these samples show that the chemical used had any effect upon the digestion. The amount of reduction in any case is inconsiderable.

Conclusions

I. It is agreed by most observers that the blood, bile, urine, saliva and salivary extract of the animals referred to in this paper contain an amylase.

II. The amount of amylase in these products in the domestic animals is slight. The saliva of the pig is agreed to have amyloclastic power that will carry the digestion of starch to the point where an appreciable amount of reducing sugar may be detected.

III. Samples of blood, salivary extract and urine from the horse, ox and sheep after having been digested with soluble starch

did not give an average reduction sufficient to warrant the statement that the amylase present in these substances carried the breaking down of starch beyond the achromic end point. The same conclusion applies to the blood of the pig.

IV. Results obtained in this work after having digested samples of bile from the ox, sheep and pig with soluble starch, did not give the results expected. We can not concur in the idea that the bile contains an amylase that is of prime importance in the digestion of carbohydrates.

V. Digesting soluble starch with saliva of the horse, salivary extract, blood, urine, or bile in the presence of 2 per cent calcium chloride does not show that reagent to increase the starch-splitting power of the amylases present.

VI. The samples of horse saliva showed a considerable reduction per cc. The amount of reduction obtained would indicate an amylase sufficiently powerful to digest starch to the maltose stage at least.

VII. The results obtained from the salivary extract of the pig indicate an enzyme powerful beyond the achromic end point produced in the glands of that animal.

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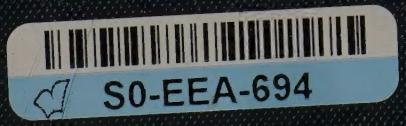
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